T. J. H.

TRANSACTIONS

OF THE

FOURTEENTH ANNUAL MEETING

OF THE

AMERICAN LARYNGOLOGICAL, RHINOLOGICAL AND OTOLOGICAL SOCIETY.

HELD IN

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TRANSACTIONS

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FOURTEENTH ANNUAL MEETING

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AMERICAN LARYNGOLOGICAL, RHINOLOGICAL AND OTOLOGICAL SOCIETY.

Held in Pittsburgh, Pa., May 28, 29 and 30, 1908.

SYMPOSIUM ON THE COSMETIC AND PLASTIC SURGERY OF THE NOSE, THROAT AND EAR.

PAPER:

THE SURGERY OF EXTERNAL NASAL DEFORMITIES.

JOSEPH C. BECK, M. D.

This subject has been and is yet, to a great extent, treated by the general surgeon, notwithstanding the fact that detail intranasal surgery is often necessary to obtain the best results. Again, a large majority of external nasal deformities are treated by charlatans because general surgeons, as well as rhinologists, often refuse and discourage treatment of the above named conditions for cosmetic purposes. This fact, I believe, is responsible for many bad results, accidents, and malformations, rather than improvements. Many such patients belong to the care of a neurologist, and should receive the strictest attention, and by all means prevent them from falling into the hands of an unskilled so-called specialist or beauty-doctor.

The various external nasal deformities that one commonly meets with may be grouped as follows:

1. Those due to marked deflections and irregularities of the septum.

Fig. 1. Twisted or Crooked Nose.

Fig. 2. Notched Nose.

Fig. 3. Squashed or Flat Nose.

2. Those dependent upon a deformity of the structures of the external nose, as the nasal bones, nasal and alveolar process of the superior maxilla, and the alar cartilages, columna, with the skin covering all the above named structures. This includes the vestibular areas.

Of the first group, one of the most frequent deformities one meets with is the twisted or crooked nose. (Fig. I.) This may vary in degree, and be to either side. As a rule, there is associated with it a hump or more or less prominence; also a dislocation of

Fig. 4. Congenital Flat Nose.

Fig. 5. Large Hump Nose.

the anterior triangular cartilage of the septum into one of the nostrils is frequently found.

Another variety of the first group is the notched nose, (Fig. II), that is, the bridge of the nose which is made up by the cartilaginous septum alone, is caved in. This is usually due to some pathological condition, as lues, tuberculosis and pyogenic infection and destruction of the cartilaginous septum, with secondary contraction.

A third variety is the squashed or flattened nose. (Fig. III.) This condition is due to the traumatic severance of the anterior triangular cartilage from the bony septum, or the latter may be also

involved in the process. This is frequently found in prize fighters, football players, or in consequence of a heavy fall on the nose.

A fourth variety, resembling very much the last named one, is that of congenital absence of the maxillary spine and underlying bony tissues of the alveolar process—in other words, the median portion of the premaxillary bones. In consequence of this deficiency, there is a complete lack of resiliency to the tip of the nose, and a distinct folding in or notched appearance at the junction of the nasal frenum and upper lip. (Fig. IV.)

Fig. 6, 7. Saddle Nose.

Fig. 8. Saddle Nose Cast.

Of the second group, namely, dependent on affection of the external structures, there are very many more varieties and the intranasal structures may be frequently associated in the changed process.

1. The large hump nose (Fig. V.) The nasal bones are massive, curved and large. X-ray pictures show a distinct increase in the thickness of bone. This deformity is characteristic of the Semitic race.

2. The broad bridge or flat nose. (Fig. VI.) In these cases the soft parts, as well as the bony framework, are flattened out.

Fig. 9. Pushed in Nose.

Fig. 10. Pushed in Nose.

Fig. 11. Pushed-in Nose.

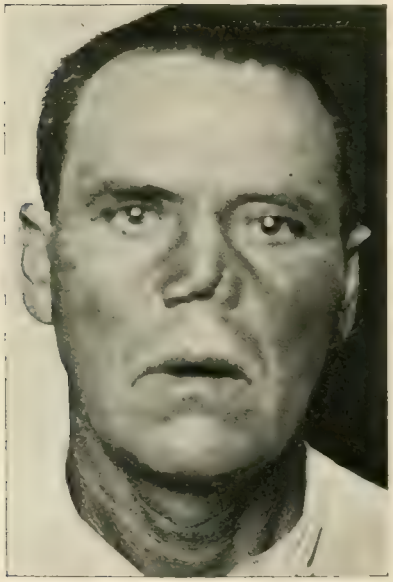


Fig. 12. Unilateral Deformity.

Fig. 14. Pound Nose.

The interior of the nose is usually very roomy and short antero-posteriorly.

3. Marked saddle nose (Fig. VII.), not due to any pathological condition. The one characteristic and objectionable thing is the much turned up tip of the nose, and very large anterior external naris.

4. Saddle nose (Fig. VIII.), due to pathological conditions, particularly in the late stages of lues, or in congenital lues. These cases are associated with, or follow closely upon, severe intranasal ulcerative processes, with necrosis of the septum, nasal bones, lateral wall of the nose. Very frequently the hard palate or floor of the nose is simultaneously affected, and a sequestration with a perforation follows. The soft parts of the external nose may also be at-

Fig. 13. Hare-Lip Nose.

tacked and destroyed. The ultimate result of this marked pathological process is cicatricial contraction, which finally causes the great deformities.

5. The so-called pushed-in nose. (Figs. IX, X, XI.) This is seen in cases where the nasal bones are not collapsed, but simply all the soft parts are affected, including all or most of the septum. Some of the cases have the skin drawn tightly across the former tip of the nose, that is, from one fossa pyriforma to the opposite. In some it is exulcerative, and the nasal mucous membrane is constantly exposed. In some the skin is drawn away in and up, making a large cavity where the protruding nose was formerly situated. The anterior nares are usually represented as two small irregular openings. The cause of such an affection is late syphilis, although lupus, rhinoscleroma and tuberculosis can cause the same trouble.

A very infectious process (furuncle) has been followed by the above named deformity.

6. Traumatic destructions and irregular deformities cannot be described, except each case by itself. It may take on any of the forms or appearances mentioned, or a combination of them.

7. Unilateral deformity (Fig. XII.) usually right, through the alar region, is frequently seen in consequence of tuberculosis, although leutic cases of that kind are quite frequent in the literature.

8. Hare-lip nose. (Fig. XIII.) This may be unilateral or bilateral. It may be a large gap right through into the nasal cavity, or it

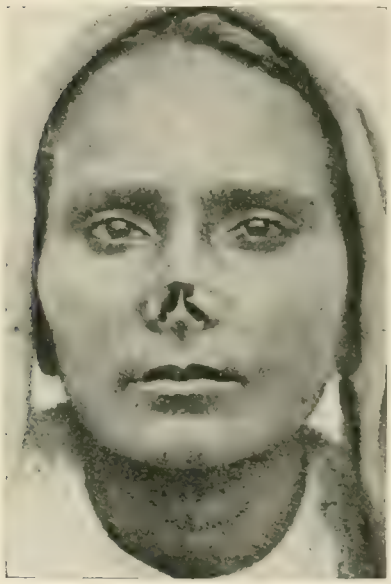


Fig. 15. Hacked Off Nose.

Fig. 16. Exulcerated Nose and Face.

may have been already repaired. While it may have been the best result the surgeon could obtain when he repaired the hare-lip, still the nose does not look absolutely normal. Usually the alae are not properly fixed, and in consequence the nostrils look broad and out of shape.

9. The pinched nose. In cases of healed-out lupus, especially of the tip and vestibule, cicatrices form in such a case as though the tip of the nose was being constantly pinched. It looks very small and out of proportion to the rest of the nose.

10. The pound nose (Fig. XIV.) (Pfund Nase). This is the large bulbous end of the nose in acne rosacea. These vary in size and degree. Small and large excrescences stud the whole surface of the nose and the neighboring portions of the face are spotted and eczematous. Cases are on record, (the author has seen one), where the pediculated tumor springing from the nose was as large as a small apple hanging by the pedicle. This is a true pathological condition of an atheromatous character, and is found most frequently in drinkers of alcoholics.

11. Hacked off nose. (Fig. XV.) A deformity very rarely seen in this country, but a common condition among the women in the orient, where punishment is dealt out in such a manner as to cut off the soft portions, including cartilages of the noses, and leaving it to heal by granulations. In sword duels and in war times, this accident has occurred. Accidentally this condition has occurred, as a plate of glass, etc., may cut off the whole, or a portion of the nose. The author knows a few cases where the latter accident has happened.

12. Exulcerated Nose and Face. (Fig. XVI.)

These are usually luetic patients, and especially in children (congenital). The process takes on the form of a granuloma, and is often mistaken for a tubercular process. The skin covering the nose, cheeks, eyelids and forehead, as also the upper lip, exulcerates; the cartilaginous portion of the external nose is fairly well preserved. In some cases bony destruction also accompanies the process. Cicatrization is very slow, and a more unsightly picture cannot be imagined, with the nasal secretion constantly bathing the face.

These illustrations of deformities are the cases out of my practice or referred to me by other physicians, and some will be shown later corrected.

I have purposely used very commonplace terms in classifying these deformities, because they are most frequently so spoken of.

Plastic surgery principles, especially when flaps are used:

1. Proper measurements of the flaps must be made as to their shapes and sizes so as to cover the defect.

2. Always allow for shrinkage of flaps.

3. Do not make the flaps too thin and jeopardize their life. When pedicles are used, see that they are not too narrow, so that plenty of blood supply enters the flaps.

4. Do not twist the pedicle too acutely and thereby strangulate the flap.

5. Thoroughly freshen surfaces of the defective area and undermine the edges properly so as to insure good union of the flaps.

6. Never use much tension on the flaps, and a counter incision in the vicinity may be necessary to relieve tension.

7. Perfect adaptation of the wound edges is imperative, and exact suturing is desirable; at the same time, do not place the sutures too close to one another, because death of the edges may take place from strangulation.

8. The finest of needles and suture material should be used. Horsehair makes excellent material. Handle the flap with great care by not using too large a rat-tooth forceps.

9. If, in the next day or two, the flap looks anemic and edematous or blue and blistered, one may have to relieve some sutures and apply warm compresses. A few small punctures with the knife in the edematous skin is at times of value.

10. Time is an important element in these operations, in that it requires a great deal of it. Do not work fast.

The indications for the correction of external nasal deformities are:

1. To establish a proper respiratory organ and correct all the pathological conditions dependent upon the imperfect ventilation and drainage. These are the cases that obstruct the anterior naris principally.

2. Cosmetic purposes. Two distinct indications may be mentioned here, namely, (a) where the deformity is of such a type as to prevent the individual from being permitted to associate with other people, owing to the obnoxious appearance, of being denied the same privileges as others, and of finding a suitable occupation. For example, I know an excellent mechanic who could not find employment, owing to his external nasal deformity. The other workers in the shop refused to work beside him or to handle any of his work. Many of these individuals will hide and veil their faces—in fact, many will remain in seclusion constantly rather than be subjects of curiosity and comment. (b) The second indication for cosmetic purposes is by far the larger number. I had intended to place this group of cases as contraindications at first, but reconsidered the matter in order to establish a point for discussion as to the limitation of correcting these deformities. I allude to the in-

dividual who does not like the shape of his nose. It is either too long or too short, flat or humped, crooked and twisted. The point is too long, broad, thick, flat, curved too much, or turned up too far. There may be a slight irregularity over the bridge of the nose or a slight depression. In fact, a number of deformities may present themselves. These are the cases which present themselves by the hundreds to the beauty doctors, and, as a rule, are spurned and discouraged by physicians. Those of you who have seen some of these cases after they were corrected by these so-called specialists, wonder if you have not acted unwisely by refusing to listen to these unfortunate individuals. I am inclined to believe that there is a slight mental unbalance, referable especially to this subject of their nasal deformities, and I am sure that every one has seen morose and melancholy individuals who were conscious of the fact that they have slightly deformed noses. The indication in these cases is possibly to refer them to a neurologist; but his counsel and treatment will not cure them. Now, since these cases occur principally among the better class of people, if one is convinced that they will carry out their way of having a slight deformity corrected, if the patient is not willing to listen to reason and leave the matter alone, may not the indication be present for us to correct these deformities, rather than permit these patients to drift into unskilled hands, especially into the hands of those who are not well versed in surgical principles, for oftentimes such patients may be fleeced out of large sums of money. It must not be forgotten that most of these individuals are never absolutely pleased and satisfied with their results, and from this standpoint I find they had better be left alone. There is, however, a suggestion to offer, and that is to have, before operation, a signed agreement that the patient assumes all the consequences so far as the cosmetic effect is concerned.

Again, I wish to be distinctly understood that I am making this indication for the purpose of discussion only, for I have heretofore always refused to correct these deformities, and will continue to do so, unless my point is well taken.

In order to correct an external nasal deformity, one should have a pretty good idea as to what shape the nose should have after correction. This is important in association with the rest of the facial and body expressions. The best guide is naturally a picture or cast of the nose previous to its deformity. However, this is

not always obtainable, and one should have a good idea of the conformation of facial expression and lines. I have made a close and careful study of this subject and find that Vanderpoel's text-book is the best suited to attain this.

The majority of the results obtainable in grave external nasal deformities, where plastic surgery was necessary, and especially in the transplantation of flaps, are anything but perfect—in fact, most of the noses that I have seen following these results are ugly. At the same time, the improvement, considering the original, especially in those cases where ulcerative processes and exposure of the nasal mucous membrane are present, is so marked as to have the gratitude of these patients without comparison. Right here, I want to say that that is principally all you do get—a great deal of work and appreciation. I mean to say that most of these cases occur among the poor. It is the persistence of labor that finally brings results, and one must never feel discouraged when, for example, he has made a fine flap and all looks well right after the operation, when he finds a few days later that the whole flap has sloughed, and the work has to be done over again. It is not rare to have ten or fifteen sittings before a plastic operation on the nose is completed.

METHODS.

The various methods of correcting external nasal deformities are principally two, namely, (1) intranasally, or intravestibularly; (2) externally.

Since the other gentlemen in this symposium are going to treat the subjects of intra-nasal methods of correcting nasal deformities as well as paraffin methods, I shall omit that portion of nasal plastic, except referring to it incidentally, and show some results following that method of procedure. The principal methods in vogue for external corrections are:

1. Indian methods,
2. Italian methods,
3. German or French methods,
4. Miscellaneous methods.

The Indian method purposes to replace the defective or absent portion of the nose by using a pediculated flap consisting either of skin and subcutaneous tissue from the forehead, or it may contain the periosteum and external table of the frontal bone. Figs. XVII and XVIII show such a method in a case of marked traumatic de-

struction of the nasal bones and loss of skin. The flap, with its pedicle severed after ten days, is seen, as is also the bone-periosteal skin defect. This latter is covered with a skin graft. When bone is not removed, or when the skin is very loose, the defect may be covered primarily by bringing the skin into approximation, as will be shown by Case I.

The Italian method purposes to replace the defect or absent nose by using skin flaps from the arm or forearm. Figs. XIX and XX show a young man who had a marked ulceration destruction of his nose and lip, following congenital lues. His left arm was prepared, and a large pediculated flap was made, consisting of skin and much subcutaneous connective tissue to give the flap considerable body and nourishment. It is sutured on a previously prepared surface on more than half of the nose. The arm is fixed and immobilized by plaster cast, and kept in that position for about ten days, during which time the wound can be observed and dressed. After that period the pedicle is detached from the arm and allowed to remain for a day or two, so that it should shrink and get firmer before it is sutured and conformed to make the nose. This picture shows well the two principal steps in the procedure.

The German or French methods purpose to cover the defect or replace the absent nose by obtaining flaps from the cheeks and other neighboring portions of the face. This method is particularly adopted for small unilateral defects, or the formation of a columna from the upper lip. The formation of the alae is a very neat procedure from the cheeks. However, for large defects or complete absence of the nose these methods are not followed by good results.

Under the head of miscellaneous methods may be mentioned the use of the finger, which is implanted and held in position by holding the arm for a week or two in a plaster cast, then amputate at the second joint, or even at the metacarpophalangeal articulation, and thus obtain a firm septum and skin cover. This method is not greatly favored.

The introduction of bone or other foreign substances, as gold, silver, hard rubber, etc., in saddle noses, as well as the exsection of bone in hump noses, will be described in connection with cases, as will also other methods of procedure.

Since there are many varieties of external nasal deformities, and each case is a law to itself, there are naturally many forms of opera-

Fig. 17.

Fig. 18.

Demonstration of the Indian Method.

Fig. 19.

Fig. 20.

Italian Method.

tions devised, consequently, I shall take up the principal deformities in presenting cases very briefly, and describe the method of operating, incidentally mentioning other methods.

Case I. Miss W., 30 years old, had lues when 20 years of age, which was not diagnosed and treated for four years. Finally there was complete loss of the nasal septum, all turbinals on both sides, and some of the lateral wall of the nose. There was a large perforation of the hard and soft palate about the size of a dollar. The uvula was lost by an ulcerative process. The external soft parts of the nose, including most of the cartilaginous alae, were destroyed

Fig. 21.

and replaced by a tightly drawn scar, leaving a small single opening in the place of the nostrils. Patient had a very thorough antispecific treatment, and was generally absolutely well.

With the above history she was referred to me by Dr. Brophy. Examination revealed a healthy looking mucous membrane of the nose (examined through perforation) and throat.

Fig. X shows her appearance at this time. The first procedure was to resect the scar and find the small remains of the cartilages of the alae. Then a columna was made out of the upper lip. I then made two flaps, one from each cheek (German or French

method) suturing these different parts together, so as to make a fairly good looking nose at the time of operation, and expecting to place within the nose some mechanical support, instead of the septum, attached to a dental rubber plate, which the patient was wearing to phonate properly, when healing took place. In the meantime, two small rubber tubes were inserted, one in each nostril, and a gauze pack to support this new formed nose. The healing was prompt and the contrivance attached to the rubber plate was introduced on the tenth day. All looked fairly well for about a month, when the flaps began to shrink, and very soon the nose looked about the same as before the operation. About three months later I used the right arm (Italian method) to obtain a large flap. The arm was thoroughly prepared and a large flap (making allowance for retraction) was made; the above retracted skin of the nose was resected and made good, substantial, freshened surfaces. We then approximated the arm and sutured most of the flap. Bringing the forearm over the head, we fixed it by means of a plaster cast so as to immobilize the whole arm. This operation was done in the summer months, and the patient suffered a great deal of inconvenience. Only a portion of the flap remained united, and made it impossible for me to complete the operation when the arm was released. In the fall of the same year I used the left arm in the same manner with very poor results. In the winter of that year I decided to use a flap (Indian method) from the forehead. This was implanted into a previously prepared defect. It healed by primary union. On the tenth day the stitches were removed, the pedicle cut loose, and properly implanted. The defect caused by the excision on the forehead was brought together and it, too, healed by primary union, although considerable tension was necessary to approximate the edges. It further required about half a dozen small operations, for instance, making a new columna bringing the alae out, so as to make good-sized nostrils. We injected paraffin below the flap to give it consistence for a tip of the nose. The end result is shown in Fig. XXI.

Case II. Mrs. E., aged 43. Has practically the same history of lues which was not diagnosed and treated for several years. The end result was complete destruction of the soft palate. The whole external nose was destroyed—that is, the cartilaginous portions, and the upper lip has multiple scars which radiate to the cheeks. Fig. IX shows the patient at her presentation. This woman was ab-

Fig. 22.

Fig. 23.

solutely dependent on her own resources and had to work. She was a fine seamstress, but could not get employment owing to her appearance. She preferred not to be operated on, if it was possible to correct the deformity without operation. Considering the situation, the fact that her general health was not very good, and the multiple hard scars that were present, I decided to use an artificial nose, (Fig. XXII), which was very satisfactory to the patient. Fig. XXIII shows the patient after the appliance is properly adjusted. This artificial nose was made by Hofman of Chicago, of flesh-colored celluloid, held by a celluloid spring and the nose-piece of her spectacles. Unless one looks at her very closely, it is not possible to discover this prosthesis, and some advise the use of actor's paste to do away with the junction of the nose with

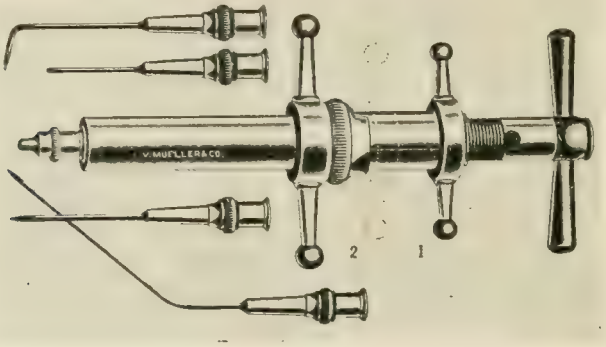


Fig. 24.

the cheek. I believe in similar cases this method of correction is preferable.

Case III. Miss M., 13 years old. Congenital lues, with marked destruction of the septum and lateral wall of the nose. End result a very marked saddle nose with the skin from the bridge of the nose folding over the inner canthus of the eyes and causing trouble with the tear duct. (Fig. VIII.)

That it was not the nasal canal that was causing the epiphora was later proven by the cure, which was accomplished by injecting paraffin. The method of procedure in correcting saddle noses or notches by means of paraffin is so well known that I do not think it is necessary to go into details. Hard paraffin is used, injected in semi-solid form by means of the author's syringe. (Fig. XXIV.) The entire saddle-shaped deformity is filled out at once

and rapidly formed into a suitable bridge. Ice is applied immediately over the injected area. Secondary correction by reinjection is sometimes necessary. Too great a quantity may be injected, thereby causing a worse deformity than before, or if one is not careful, it may be injected and allowed to spread in a wrong direction. Again, from too great a quantity necrosis of the skin may result. Infection, hematoma, hemorrhage and embolism belong to the rarest complications. Injections in series of several sittings are followed by a lumpy correction. By observing the proper technic laid down, namely, perfect asepsis, perfect instrumentation, assistant to prevent spreading in a wrong direction by compressing the skin at the side and upper limit of the bridge of the nose against the underlying bone, avoiding the entrance of the needle into a large artery or vein, usually injecting near the middle line, and inserting the needle separately from the syringe, if no bleeding occurs through the needle, the complications of hematoma, hemorrhage and embolism will be avoided.

Good judgment is the only criterion against injecting too much paraffin, and if one should do so rather than through a small incision, one can remove parts of paraffin for some time after operation without any evidence of scars. A mistake made sometimes is to inject the paraffin into the skin proper, rather than below it, which invariably is followed by death of the epidermis. If one wishes to inject in series, then he had better use the method devised by Onoli and others, using semi solid paraffin that can be moulded. Some interest has been awakened as to the termination of the paraffin, and many experiments have been carried out to determine the same. Kirchner states that soft paraffins are absorbed, but hard paraffins are not. It is stated that new connective tissue forms as a meshwork within the substance of the paraffin. Histological examination makes the bolus appear like a tumor, some say resembling a sarcoma, and it is spoken of as a paraffinoma. The result after the procedure just described above in the present case is shown in Fig. XXV.

Case IV. A young girl, 7 years of age, with a marked ulcerative process of the entire external nose, including the bony framework, eyelids, parts of cheek and upper lip. (Fig. XVI.) The pathological diagnosis was tuberculosis and syphilis. No local or general treatment had any effect whatever. So far as the other parts of the ulcerative areas were concerned, not included in the

nose, they were covered by skin grafts and neighboring flaps, and the nose was left to the last. Several methods were used, as described in Case I., including the use of the finger. This method is as follows: Take the middle finger of the left hand, remove the nail and matrix, make an incision into the skin and underlying connective tissue in the middle line along the entire palmar surface, beginning at the tip and carrying it to the phalango-metacarpal joint, dissecting these flaps laterally. Freshen the point of the finger by exposing the bone proper and make a slanting surface which fits later into the region of the root of the nose. We next

Fig. 8.

Fig. 25

prepare the nasal field, as mentioned above, also freshen the surfaces along the nasal processes of the superior maxilla. The tip of the finger is then placed into the root of the nose, and there held by suturing first the periosteum, and then the skin to these structures of the forehead. Then the skin flaps are united to the surfaces of the nasal processes of the superior maxilla. The arm is then supported and fixed to the head by a plaster cast. This precise procedure was carried out in this case. After about ten days, during which time the field of operation was daily exam-

ined and dressed without the slightest manipulation, it was found united and in good condition. The next step in the operation was to disjoint the finger at the phalango-metacarpal joint, and so shape the finger at this point as to form a good tip of the nose. It was necessary to trim off some bone in order to make nostrils.

This end of the finger is placed into a previously freshened premaxillary area, and there fastened with a wire suture. The remaining skin of the finger is used to best advantage to cover raw surfaces and the formation of alae and nostrils.

Fig 16.

Fig 26.

This method was also followed by failure, and finally the last attempt was made, which proved to be successful, namely, the Indian method was used in making a flap from the forehead. The case required a great deal of attention and labor. It was practically two years of dressing and treatment, but the result was satisfactory.

I would like to mention one fact in this case, and that is, that this child was kept by its own people in the barn away from the rest of the family. It was the child of ignorant parents who believed themselves cursed, and did not want the neighbors to know

anything about its existence. A country physician discovered the case and sent it to Chicago for treatment.

Permit me to mention a similar method used, but which is only applicable when the nose has skin covering it. In those cases the skin is removed from the dorsal surface of finger, the latter is then pushed into the nose, and its tip fastened into the root of the nose. In this way the finger is used as a septum. The palmar skin covered surface acts as though it was covered with mucous membrane of the interior of the nose. (J. M. Finney.)

Case V. Markedly twisted nose to the right, with the anterior triangular cartilage protruding out of the right nostril. Patient

g. 27. Cast Before Operation

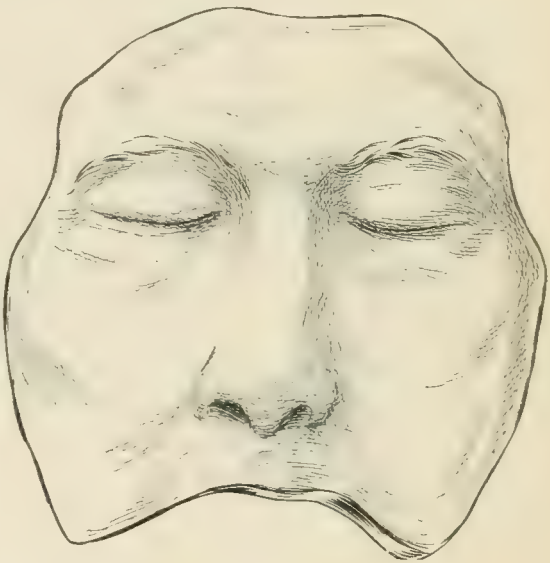


Fig. 27. Cast After Operation.

complained but little of this nasal obstruction, although he could not breathe freely through either nostril. What he wanted was a straight nose. I informed him that I would remove the obstruction, which would very likely correct most of the deformity, and he readily acceded to the proposition. I resected the anterior dislocated cartilage on the right side, and did a submucous operation from the left, leaving a layer of cartilage at the frenum. I had a most excellent physiological and cosmetic result, as Fig. XXVII shows (casts). Refused publication of photograph.

Case VI. Miss F., 20 years old. Has a very broad bridge of nose, and a markedly deviated septum. While a small child, sitting on a high chair her father, while intoxicated, pushed her head against the edge of the table, fractured and pushed her nasal bones inward. (Fig. VII.) Operation. I made an incision within the alae on each side, so as to reach the free border of the nasal bones, then bluntly dissected below the skin as far as the root of the nose. Next I introduced a pair of Adams' modified osteoclastic forceps and broke the nasal bone union in the middle line in such a way as to form a sharp edge over the bridge. Then I drilled a hole externally through the nasal bones and passed a 3-

Fig. 28.

Fig. 7.

inch silver peg. I covered the bridge of the nose with a layer of gauze, so as to avoid pressure necrosis from the now applied silver wire in the shape of a figure eight loop from one protruding point of the peg to the other. This peg and wire procedure prevents the collapse of the nose. In addition, two Frankel's semi-solid splints were introduced along the side of the septum to support the nasal bones from within. Small drainage silkworm strands are introduced into the alar incisions. The case made an uneventful recovery, with the exception of considerable reaction which, however, cleared up. The pegs were removed on the fifth day, and the openings were the only points of infection. Fig. XXVIII.

shows you the final result. (Patient refused to have picture taken.)

Other methods for correcting such a deformity in vogue are to make a longitudinal incision along the bridge of the nose, incising the periosteum and peeling back. Then over a properly prepared anterior tibial surface an incision is made, and the sharp edge of the anterior border of the tibia is chiseled off and shaped; the periosteum is to remain attached to the bone splinter to insure its life. The nasal bones are then scraped, or a bur may be used and the bone splinter introduced. The nasal periosteum is then drawn well over it and sutured with catgut. The skin is then sutured care-

Fig. 29.

Fig. 30.

fully, using various methods. The Halstead subdermal suture makes a good one, as there are no stitch holes possible. Dr. Carl Beck of Chicago suggests making a skin graft flap before incising the skin, which, when the operation is completed, serves to cover up the cut.

Another method is to introduce foreign substances, like aluminum, silver, platinum, and gold plates, but all these methods are losing favor. Of course, paraffin injections are very serviceable in these cases.

Case VII. Mr. H. Large hump nose. Operation was done purely to correct the deformity for cosmetic purposes. His deformity

caused his hypersensitiveness to grow to a degree of melancholia. He shunned society—in fact, he did not feel like going to work. He is a salesman by occupation.

Operation. A transverse semilunar incision, with the convexity upwards, was made, beginning at the nasal process of the superior maxilla on one side, and carried to a corresponding point on the opposite side. The skin and periosteum were peeled down as far as possible to the lower limit of the hump. By means of Killian's wedge-shaped chisel the hump was removed, great care being taken not to open through the mucous membrane into the nasal cavity. In spite of care, it occurred in two places. The periosteum was

Fig. 31.

drawn over and sutured carefully, as was the skin. Patient made an uneventful recovery. No drainage was used. An attempt was made to bring the bones together after the hump was excised, but it was impossible.

The incision is, at times, made longitudinally along the ridge, with equally good results. Of course the incision I made is practically hidden by the nose piece of the spectacles. Intranasal methods are at times used to correct this deformity.

Case VIII. Miss I. Congenital absence of premaxillary bony ridge, with the characteristic flat tip of nose. (Fig. IV.) Purely for cosmetic purposes, the patient presented herself for operation.

Operation. An intranasal incision was made at the floor of the nose, beginning at the entrance of the nose, and carried about one and one-half inches backward. Blunt dissection was made of the muco-perichondrium and osteum, chiseled off a spiculum of bone from the floor of the nose; made a pocket below the columna loosening its attachment to the premaxilla, turned this bone spiculum into this pocket; brought back the muco-perichondrial flaps, and held them with packing. On the third day I removed the packing. Healing took place, and considerable improvement followed. Fig. XXXI. shows the condition after operation, and some subsequent paraffin injection.

Case IX. Mrs. X., colored; about 45 years ago she had some trouble in her nose, which was followed by a caving in of the whole tip, especially the left side, and finally it grew fast in that shape. There is no history of syphilis obtainable, and the woman has three healthy children. Examination reveals a complete destruction of the cartilaginous and bony septum, but the nasal bones and lateral margins of the bony nasal framework are intact. The cartilaginous alae are preserved, however drawn inwards, especially the left side, and firmly held, with the skin of the tip of the nose which is intact, to the floor of the nose. The nasal frenum or columna is also preserved. Fig. XI shows the condition as above described.

OPERATION.

Under general anesthesia by the spray-vapor method. I mention this especially because it was the most satisfactory narcosis I had ever given for a similar operation. The following steps were performed: Packed nose. Severed the adhesions of the drawn-in nose to the floor, thoroughly severing all attachments to the lateral margins of the bony framework, as well as the free borders of the nasal bones, as far up as the root of the nose. All these procedures were made within the nose, no incision being made externally. This permitted drawing out the whole tip prominently. I now scraped very thoroughly the nasal bones, just below the skin, and also the floor of the nose right behind the columna. This was done in order to obtain fresh bone surfaces for the implanted

bone septum. Packing the nose, I next made an incision over the previously prepared anterior border of the right tibia for about four inches up to the periosteum. A two and one-half inch piece of bone was now excised with the periosteum attached, using the sharp ridge of the tibia as the future nasal septum. The shape of this splinter was triangular, with the base downward, to be implanted at the floor of the nose and the apex on top of the nasal bone. The periosteum of this splinter was now in contact with the subcutaneous connective tissue of the bridge of the nose. A suture was now passed through the periosteum of the splinter, right back of the column of the nasi, and allowed to come out

Fig. 32.

below each nostril through the upper lip, and close to the frenum, a pad of gauze was placed externally over the frenum and the suture tied. This was done to hold the splinter in position to give prominence to the tip of the nose, the gauze packing is now completely removed, and two large Asch tubes are introduced, these being held from falling back into the nasal cavity by two threads fastened by adhesive plaster to the cheeks. No dressing was applied. The patient recovered from the operation, and Fig. XXXII shows her after operation.

Case X. Mr. S., (Fig. XIV.), mild form of pound nose, acne rosacea. There is a general overgrowth of the soft parts of the external nose. A number of studded areas are present, with a central breaking down. The skin is generally eczematous and deeply red. Distinct large venules are to be seen everywhere over the nose. This process extends to the forehead and side of cheeks, The man is not a marked drinker, and the withholding of liquor has had no effect on it. The treatment in this case is very satisfactory by means of the high frequency currents and electrolysis. After a few sittings the nose became paler and smaller, as shown in Fig. XXXIII.

Other methods of treatment in these cases have been to use electrolysis, electric needle punctures (galvanic current), electrocautery, severing the small blood vessels surgically, multiple incision, and the operation of decortication and skin grafting. This last procedure is only warranted in very marked cases. It simply means to remove all the pathological tissues and cover with skin graft.

Case XI. Baby M., 1½ years old. Had an operation for hare-lip shortly after birth. The result was very satisfactory, but the nostril was not perfect. Its greater diameter was transversely rather than antero-posteriorly.

Operation. This consisted in freeing the ala from the cheek and drawing it closer to the columna, so as to conform to the shape of the opposite nostril. Then the cheek was slightly loosened from its attachment at the fossa pyriforma and drawn over to the new fixed alae in its new position and sutured. Healing by primary union occurred, and the result was good. A small Asch tube was worn in the nose for a few weeks, three hours daily, in order to obtain and preserve the proper shape of nostril. In double-sided hare-lips a similar procedure may be necessary. No photograph obtainable.

Cases XII and XIII. Both these cases have practically the same form of deformity, and the same method of procedure was used, so I will consider them together.

Mr. W., medical student, has been suffering from chronic nasal obstruction, which was due to a markedly deflected septum. This was operated upon by the submucous method. An unusual and disagreeable complication resulted, namely, a constant bleeding. Patient was sent to a hospital, where inexperienced men in this line

of work attempted to control this bleeding, the patient's own physician (a specialist) being out of the city at the time. According to the statement of the student, the internes used all sorts of remedies by spray, swab and pack, as H_2O_2, adrenalin, ichthyol and iron, without controlling the bleeding. I saw the patient one week after operation, and there was still bleeding from within the flaps. The operation was perfect, but the flaps were not coapted, and a big hematoma was situated between them. I removed this, coapted the flaps, and kept in this position by firm packing in both nostrils. Hemorrhage ceased, and on the third day the packing was re-

Fig. 33.

moved. There was no more bleeding, but after that the nose was stuffy for some time, but finally cleared up and good breathing space was obtained. Six weeks later the patient came back with a sunken-in nose, as shown in Fig. II, the notched nose. This notch is situated just below the free border of the nasal bone, and more on the left than on the right side.

Operation consisted in injecting a small amount of hard paraffin, subcutaneously, to fill out the notch, with a good result.

M. H., 7 years old, received a blow from another boy on his nose, and his nose swelled inside, so that he could not breathe through

it for about a week. Then his mother noticed a discharge from his nose, and he was well again.

Three weeks later she noticed suddenly a notch in his nose, between the tip and the hard portion.

The diagnosis was probable abscess of the septum, following traumatic chondritis. There was secondary wasting of the cartilage, with contraction of the cicatricial tissue, and sinking in of the septum. Hard paraffin was injected with satisfactory results.

So far as the numerous external nasal deformities, such as moles, blemishes, wrinkles, scars, and the various insignificant and often imaginary troubles are concerned, I have nothing to say at present, because I have already expressed myself in the first part of my paper. Tumors and other pathological conditions not mentioned in the article will be dealt with by the usual surgical principles and cosmetic surgery.

As to the literature on the subject, it is very exhaustive, and to repeat it would add little value to this article. But I want to mention and to present here a little original book of Tribhovandas Motichaud Shah L. M., assistant surgeon and chief medical officer of Junagadh, presented to Dr. Carl Beck. In it are detailed descriptions of 100 rhino-plastic operations performed by himself. Two principal methods were used, namely, flaps from cheeks and forehead were used. The latter gave the best results.

A more modern reference is the one of Dr. John D. Roberts, entitled "Deformities of the Face, with Special Reference to Nasal Plastics." Some of his methods are very unique and appear to have given him good results.

Such men as Tagliacozzi, Langenbeck, Ollier, Senn, Keegan, Roe, Bunger, Dieffenbach, Fraefe, Blaudin, Serre, Jobert, Zeis, Warren, Mutter, Pancoast, Post, Buck, Andrews, Prince, Seymanowski, Reverdin, Krause, Thiersch, Wolfe, Joseph von Eiselsberg, J. B. Murphy and Carl Beck, Chicago, must be recognized as great workers in this line of plastic surgery.

PAPER:

THE CORRECTION OF NASAL DEFORMITIES.

JOHN O. ROE, M. D.

In compliance with the request to take part in this discussion on "The Correction of Nasal Deformities," I shall confine my remarks to a brief discussion of the subcutaneous method, which does not involve injuries to the skin, except in those cases where it is necessary to remove unsightly scars that would remain a pronounced blemish to a nose, however comely in size, shape and general conformation it might otherwise be.

There is no class of operations that demand in every case a more careful preliminary study of all the conditions presented—not only in respect to the abnormal state of the tissues to be operated upon, but also in respect to the possibility of obtaining the desired surgical result than those operations required for the correction of nasal deformities.

In studying the etiology of nasal deformities it must be borne in mind that such an abnormal condition may be due to a great variety of causes. It may be due to an abnormal development of the nose, an overgrowth amounting in some cases to an elephantiasis; or it may be due to an unequal growth of the two sides of the nose, or of the two sides of the laminae of the septum, distorting both the septum and the nose. The distortion may be the result of an extra or intra-nasal neoplasm, or, as is frequently the case, of a traumatism with a definite history, or presenting a pronounced mark of the injury. It may be of constitutional origin with a leutic history, presenting the definite specific manifestation shown in the excavated, shrunken and caved-in nose that results from the destruction of the bony or cartilaginous framework, or perhaps of both, and sometimes shown in the dactylitic condition occasionally seen. The deformity may be the result of lupus that has been arrested, leaving scars and deformities, which must be carefully

differentiated from leutic manifestations. The abnormal condition also may be of a tuberculous nature, associated with similar manifestations in other parts of the body, or finally, it may be caused by ulceration or by a growth of a malignant nature.

Having ascertained the etiology of the deformity, we can then intelligently examine the nose to determine the exact nature of the deformity, the condition of the tissues and the anatomical structures, both external and internal, that are affected by or that enter into the deformity.

After this careful study of the cause and nature of the deformity it is necessary to consider the possibility of obtaining the result desired in its correction and to advise intelligently as to the desirability of the operation and the probability of its being a success. It may be said on general principles that the correction of a deformed nose, in which so much is involved to both the patient and the surgeon, may be likened to matrimony, which "should not be entered into unadvisedly or lightly, but reverently, discreetly and advisedly."

In all cases in which the deformity of the nose is associated with or caused by a constitutional condition, such as lues, no attempt to correct the deformity should be made until the constitutional condition has been most thoroughly treated and all manifestations entirely removed.

Without this precaution being taken the operation would be quite sure to be a failure, the disease would be excited to a greater local activity, the healing of the wound prevented, thereby resulting in greater destruction of tissue from abscess or ulceration and an increase in the deformity. When the constitutional disease has been completely arrested and all local manifestations quiescent for a considerable time (for say six months) in most of such cases little or no difference from non-specific cases will be seen in the healing of the wound, if the blood supply has not been cut off by cicatricial tissue, and if care is exercised during the operation to interfere with it as little as possible. These specific cases, however, at best must be regarded as an uncertain quantity, and will sometimes disappoint us or "go bad" when least expected.

If the deformity of the nose is found to be associated with a local disturbance inside the nose, obstructing the passages, we should invariably remove or correct this local condition, whether it be a deviation or thickening of the septum, enlargement of the

turbinates, a polypoid or other growths, or even adenoids and large tonsils.

To preserve perfect nasal respiration is of the utmost necessity, not only to the health and comfort of the patient, but to the satisfactory correction of the nasal deformity.

Having determined on the advisability of the operation, the operation itself must now be carefully studied as related to the condition and position of the tissues, the relation of the different parts of the nose to one another, and the relation of the shape of the nose to that of the face.

In studying the normal nose of different types of faces we find that the noses of people having broad or eurygnathous faces with prominent malar processes and orthognathous jaws tend

Figure 1.

Figure 2.

toward the Grecian in outline; and as this type becomes more accentuated the nose becomes more flattened in the center and thickened at the end, with more expanding wings; when, however, the shape of the jaws merges into the prognathous type, the nose becomes Roman in form.

People with narrow faces, small malar processes and orthognathous jaws have narrow, thin and aquiline noses, the aquilinity increasing as the jaws incline toward the prognathous.

The narrow face, undeveloped and retrousse nose, prognathous upper jaw and projecting incisor teeth are most distinctly the mark of an interference with or a distortion of development due to mouth breathing in infancy and childhood so often caused by

enlarged tonsils and adenoids and other naso-pharyngeal obstructions.

In correcting a nasal deformity and reconstructing a nose careful attention must therefore be paid to these various conditions.

While symmetrical relations of the different portions of the nose to one another are of the greatest importance, the symmetrical relation as to the size and shape of the nose to the general contour of the face must also be carefully considered, in order to approach the ideal from an artistic standpoint. If we superimpose an aquiline nose on a broad face with pronounced malar processes we have an incongruity quite as pronounced as putting a small Grecian nose with a low bridge on a narrowly constructed face, as is

Figure 3.

Figure 4.

shown in three or four of the illustrations. The aquiline nose in figure 2, which is very becoming to the young man with a narrow face, would ill become the broad face of the patient in figure 14 or 18; and the comparatively small Grecian nose in figure 13 or 16 would very seriously detract from the character of the face in figure 2.

A complete description of the technique is scarcely possible, for the reason that in no two cases are the same conditions present requiring the same procedure. Roberts, in his lectures on "The Surgical Treatment of Disfigurements and Deformities of the Face," very truly says:

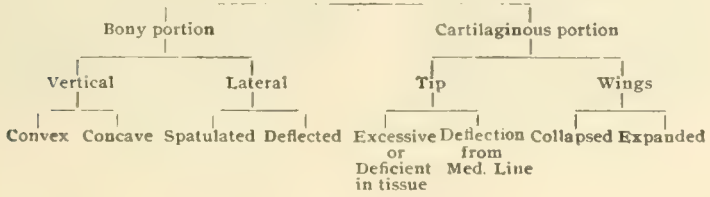
"It is difficult to give definite directions for geiloplastic procedures, because the surgeon must be governed by the peculiarities of the case under observation. Almost every time one operates

the method must be varied to meet the requirements. Often, indeed, the necessities of the case determine the line of incision only after the operation has been commenced."

This is equally true of subcutaneous plastic work. On general principles it may be said that the first step in the operation is to carefully raise the skin without injuring it from the deformed portion, the incision being made from the inside of the nostril anterior to the deformity, and the redundant portion cut away or placed in the depressed portion, so as to make the nose symmetrical and give it the shape we desire. Except in very large noses, as in figure 1, it is rarely necessary or desirable to remove any portion of tissue or even bone, for there is generally a correspondingly depressed portion that requires filling up to give the nose the proper shape. This is particularly the case where the cause of the deformity is traumatic, when we simply have a displacement rather than a destruction of tissue, which should be restored, so far as possible, to its former position.

In 1898, in an article on this subject, which I read before the British Medical Association, I classified nasal deformities as follows:

DEFORMITIES OF THE NOSE.



I here reproduce this classification for reference, as it is the one I use, and the one followed in the report of the following cases that I have selected to illustrate the different points of the technique of the subcutaneous method.

Case 1. Convex vertical deformity of the bony portion of the nose. Mr. J., aged 23, injured his nose six years before in a fistic encounter, which caused an exostosis of the nasal bones. The end of the nose was abnormally long, due to an elongation downward of the triangular cartilage, shown in figure 1. On examination there was found also a deviation of the osseo-cartilaginous portion of the septum to the right, obstructing this nostril, the left

nostril being greatly narrowed by a marked projecting of the nasal process of the superior maxilla.

The first thing done was the straightening of the septum and the removal of sufficient of the nasal process of the superior maxilla to render the nasal passages entirely free. The exostosis of the nasal bones was then removed by carefully sawing them off on a line, so as to leave the dorsum of the nose straight, having first elevated the skin from this region through an incision on the inside of the alae. As there was no depression to be filled in, the portions that were sawed off were removed. When completely healed, the elongation of the lower end of the triangular cartilage was resected and the ends strapped back in place, which when healed gave the most excellent result shown in figure 2.

Figure 5.

Figure 6.

Case 3. A concave vertical deformity of the bony portion of the 40, injured her nose three years ago last July by falling from her bicycle. The front wheel stuck in a rut and threw her over the handle bar, her nose striking a stone. When the swelling had subsided she experienced much difficulty in breathing through the nose. One year later this obstruction, in the form of a bone, was removed, evidently a portion of the deviated septum, which resulted in the sinking-in of the dorsum of the nose. Six months after this a friend of mine in another city, who believes in the efficacy of wax, filled this depression, or rather, overfilled it, with paraffin, with the result as shown in figure 3. In addition to this marked bulging of the dorsum there was a pronounced redness, which still further disfigured her nose.

In the correction of this deformity several operations were required. First, the tissue containing the paraffin was removed sufficient to bring the dorsum of the nose down nearly to a straight line. This was done subcutaneously through an incision on the inside of the ala. When this had healed, three weeks later, the distorted end was put in shape by raising the skin and filling in the depressed portions with flaps from the elevated portions. The tissues healed tardily where the paraffine had been injected, and later developed a tendency to swell when the external pressure from the dressing was removed, and after a time it became so enlarged that it was found necessary to remove some of this tissue to compel the nose to heal in the desired shape, as shown in figure 4.

Figure 7.

Figure 8.

Case 3. A concave vertical deformity of the bony portion of the nose, the result of an injury. Miss M., aged 25, fell from the second story window upon a piazza railing, when five years old. She was at the time leaning out of the window and fell head foremost, striking on her nose. The nose at the time became much swollen, but as no physician was at hand she was attended by a nurse, and no attempt was made to replace the fractured fragments of the nasal bones. When the swelling subsided the bridge of the nose was found to be very flat, almost on a line with the face, but as it was thought nothing could be done to relieve the disfigurement no further attention was given to it. Figure 5 represents the condition of the nose at the time I saw her, February 22, 1906.

On examination the nasal bones were found displaced outward and downward, leaving two well marked ridges on either side. The end of the nose was abnormally large and the wings expanded. The nasal passages were very much obstructed by ridges and thickening of the septum, that resulted from the impaction of the perpendicular plate of the ethmoid, and by marked turbinated hypertrophy on both sides.

Before correcting the deformity of the nose it was necessary to render the nasal passages free by a reduction of the turbinates in the usual way, and the submucous resection of the septal excrescences. The external deformity was then corrected by elevating the skin from the depressed portion through an incision made

Figure 9.

Figure 10.

from the inside along the upper border of the lower lateral shield cartilages. When this was done the periosteum was raised about ten millimeters outside of the displaced nasal bones. Projecting portions of the nasal bones were then sawed off and put in the medial line, together with the periosteum that had been elevated from the bone just outside of them. These were carefully held in place by an external dressing, so as to maintain them in this position until union had taken place. After the parts had thoroughly healed, the remaining depression below was filled in with the redundant tissue in the end of the nose, which was carefully cut into a flap and turned into the depression. This was also held in place by an external dressing until thoroughly healed, which left the nose in the condition shown in figure 6.

Case 4. This is an exceedingly interesting case, involving both a serious concave vertical deformity and also a deflection of the bony portion of the nose. The deformity, as shown in figures 7 and 8, had resulted from being thrown from a carriage by the breaking of the forward axle, and in the fall striking the nose upon the sharp edge of a tramway rail, causing a most frightful injury to the face, the loss of the sight of the right eye, together with a concussion of the brain, which was so severe that life was despaired of for several days after the accident. The injury to the nose completely severed the lower portion of both nasal bones, flattening the lower depressed portion completely to a level with the face, leaving the stump of the nasal bones projecting above like a ledge, as shown very clearly in figure 7, and the fearful scarring

Figure 11

Figure 12.

that resulted when the wounds healed is shown very well in figure 8. The septum was also so distorted to the left as to completely occlude the left nostril, and the knocking inward of the center of the nose had caused the end of the nose to be tilted upward and to the right, as is also shown in figure 8. As the patient formerly had quite a large nose, the end therefore appeared by comparison with the remaining portion abnormally large.

Several operations and much careful attention were required in the correction of this deformity. The first one consisted in the raising of the skin from the central portion of the deformed part through an incision from the inside of the ala, which, owing to the firm attachment of the skin in many places to the parts below, at

the site of the scars, proved to be a very difficult procedure. When this was done, a portion of the projecting nasal bones was sawed off through this incision with a nasal saw and placed in the depression below. In order to be certain of the bone's uniting quite firm pressure by the external dressing was necessary in order to keep the parts in firm coaptation. When the parts had healed from this operation, after about three weeks, the skin was raised from the lower end of the nose and also from the dorsum of the depressed portion in the center, and a flap made from the tissue in the end of the nose and turned into the portion still depressed, which elevated the center of the nose still farther. When this had healed a flap was turned in from the ridge of the tissue on the left

Figure 13.

Figure 14.

side of the nose, and likewise from the right side, and in this manner the nose was built up until we had the symmetrical result shown in figures 9 and 10.

The result in this case was highly gratifying, but required much patience, both on the part of the patient and the operator. The only disfigurement then remaining was the right eye, which was lost at the time of the accident. This, however, was rectified by the insertion of a glass eye by his oculist at his home, who had referred the patient to me.

Case 5. A. S., aged 34, a resident of Helsingfors, Finland, was kicked in the face by a horse when a year and a half old, fracturing the nasal bones and driving them inward. On examination the nose showed a concave vertical deformity of the bony portion

of the nose, with a scar on the left side of the nose over the nasal bone, which drew the left ala up considerably above the other. The septum was irregular, with a large enchondroma on the left side.

In correcting this deformity the interior of the nose was first put in order, and the enchondroma removed by a submucous resection. The external deformity was corrected by carefully raising the skin from the dorsum of the nose through an incision just inside the ala, near the tip of the nose. The skin in this case was firmly attached to the nasal bones at the seat of the injury, but by thus freely liberating it the left ala was brought down on a line with the right ala. A flap was then made of the redundant cartilage at the end of the nose and turned up into the de-

Figure 15.

Figure 16.

pressed portion between the eyes. The triangular cartilage just above the tip of the nose was then sufficiently liberated by a beveled incision from above downward, so that the end of the nose could be brought down, making the lower end of the nose almost horizontal. The nose was then held in place by a metallic splint, formed to the desired shape of the nose, which was held in place until union of all the parts had taken place. The nose was then comparatively satisfactory, excepting two or three minor defects, that required slight operations, and when these were healed the nose was left in a highly satisfactory condition. Figures 11 and 12 show the nose before, and figures 13 and 14 after the operations.

Case 6. A young man, aged 28. There was in this case deficiency in tissue of the cartilaginous portion and a lateral deflection to

the right of the bony portion, as shown in figure 15. There was also a marked expansion of the alae on both sides. The deformity had resulted from a fall when a child, before he could remember. On examination of the interior of the nose the septum was found slightly deviated to the right, with a knuckle of cartilage obstructing the right nostril. The left passage was much obstructed by a large posterior inferior turbinate hypertrophy. The operation for correcting this deformity was quite similar to the previous cases: the skin was raised from the deformed part, the marked thickening on the left side of the nose above was sawed off, and the portion removed placed in the depression below. The deformity of the lower third of the nose was corrected by taking

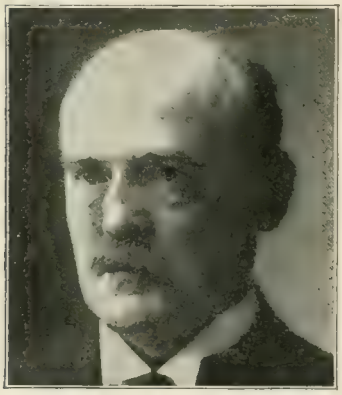


Figure 17.

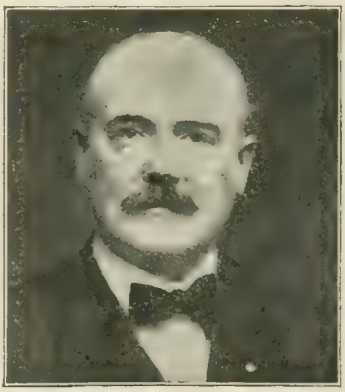


Figure 18.

off the elevated portion and turning it in by a flap operation into the depressed part. When this had healed the dorsum of the nose was quite straight and symmetrical, but the alae were still abnormally spread out, owing to an unusually wide attachment. This condition was rectified by severing the lower attachments of the cartilages subcutaneously with a slender knife, similar to a tenotomy knife. When this was done the attachments of the alae were carried inward and stitched in the position desired by means of the writer's intra-nasal suture needles, horsehair being employed for the suture, and when healed the nose was left in the condition shown in figure 16.

Case 7. W. H., aged 40. This case represents a condition we often find resulting from traumatism, combining two or three

different deformities in one nose. In this instance we have a depression of the upper cartilaginous portion of the nose, and a convex vertical deformity and lateral deflection of the bony portion, as shown in figure 17,—the result of an injury from a fistic encounter with some boys when ten years old. He has had since the injury much difficulty in breathing through the nose. On examination the left nasal bone was found displaced to the left, together with the upper dorsal cartilage. The triangular cartilage was excessively thickened, which had resulted from the crushing of the dorsum at the time of the injury. The tip of the nose also was much enlarged.

In correcting this deformity it was necessary first to reduce the thickening of the septum by resecting a portion of it by the sub-

Figure 19.

Figure 20.

mucous method, so as to render the nasal passages free. The external deformity was corrected by first elevating the skin from the deformed part, and turning up a portion of the redundant tissue in the end of the nose by a flap-operation into the cartilaginous depression. A portion was then removed from the projecting nasal bone, and also placed in the depression below. The nose was then dressed with a supporting splint on the outside in the form of a saddle, and held in place until healed. Two minor operations were required to level some slight irregularities that remained, when the nose was left in the condition shown in figure 18.

Case 8. Concave vertical deformity of the bony portion of the nose and a marked thickening of the cartilaginous portion. Miss

Y., aged 21. When six years of age, while ill with an attack of tonsillitis, she attempted to go down stairs, but was so weak that she fainted and fell the whole length of the stairs, striking on her nose. The nose became very much swollen and infected with erysipelas, which extended over her whole face. At the end of two weeks the erysipelas had subsided, leaving the nose almost completely flattened in the center.

At the time I saw her the nasal bones were flattened and dislocated outward. The sesamoid and upper shield cartilages in the dorsum of the nose had been destroyed, while the tip of the nose and alae were large and very much expanded, as shown in figure 19. The operation for the correction of this deformity consisted

Figure 21.

Figure 22.

in dissecting up a portion of this redundant tissue in the end of the nose and turning it back in the form of a flap into the depressed portion. The incision in this case was made just inside the border of the alae, raising the skin from the part that was to be dissected up and also from the depressed portion. When this was done the parts were held in place by a saddle splint on the top of the nose, free drainage being allowed from the inside. When the parts had healed and all the swelling subsided, the nose was not quite perfect, requiring a second operation similar to the first to raise the center to the desired height, and also to depress the tip and make the nose symmetrical, as shown in figure 20.

Case 9. Excessive development of the tip of the nose with an enlargement and expansion of the alae, shown in figure 21. Mrs. W., aged 30. In this case there was no history of trauma-

tism. The exuberant growth of the end of the nose had evidently resulted from persistent turgescence caused by intra-nasal pressure and obstructed nasal passages due to marked enlargement of the middle turbinates and anterior ethmoid cells, and also to lower posterior turbinate hypertrophy. Preliminary to the correction of the external deformity, the nasal obstructions were carefully removed, as in the previous cases. The enlarged ethmoid cells were obliterated by submucous resection of the inner bony wall, and the passages were maintained free by careful packing with iodoform gauze until healed. The external deformity was corrected by cutting flaps of the redundant tissue at the end of the nose and turning them back along the bridge, after having elevated the skin over this region. When this had healed the inflated and

Figure 23

Figure 24.

bulging condition of the shield cartilages was reduced by carefully incising the cartilages from the inside and strapping and holding them in place in the desired position. When this was done the nose was found to be too long in proportion with the rest of the face. This was rectified by the removal of a triangular piece of the septal cartilage and suturing the parts back in place, which left the nose when completed as shown in figure 22.

Case 10. Congenital enlargement of the entire nose, shown in figure 23. Miss M., aged 23, had had from infancy an unusually large nose. This deformity greatly annoyed her, so much as to be a positive hindrance to her in her dramatic work. The mode of reducing the size of the nose consisted first, in sawing off

a portion of the nasal bones and paring them off, subcutaneously, to lower the nose to the desired dimension. After healing, the skin was carefully raised from the lower portion of the nose and portions of the shield cartilages were resected. The nose was then carefully strapped, so as to hold it in place in the desired shape and held there until completely healed, leaving the nose as shown in figure 24, which appears as a fine symmetrical nose, being several sizes smaller than before the operation.

In the correction of nasal deformities by the subcutaneous plastic method, quite as much care must be exercised as by the external skin flap method. If too much tissue is taken from any particular part a depression will result; if too much is put in a hump will be left. If the skin is injured an undesirable scar or redness or other disturbance of the skin will result.

Quite as much patience and perseverance is required as by the other method, for subcutaneous wounds are quite as slow to heal as external wounds, and frequently more so, owing to the disturbance in the circulation often required by the quite extensive cutting we are sometimes obliged to do. It is on this account that we often have persistent swelling of the nose on removal of the external dressing for some time after the tissues are apparently healed. In these cases it is absolutely necessary to wear the dressing with sufficient pressure to keep this swelling down until the parts have become so healed that this tendency has completely disappeared.

In some instances patients have become so impatient to be freed from their nasal dressing that I have been induced to remove it prematurely, and later they were rewarded for their impatience by being obliged to have another operation which a little longer wearing of the dressing would have avoided. As I have said before in previous papers on this subject,* the reward of patience and perseverance in these cases is the satisfaction of success on the part of the surgeon and a perfect nose on the part of the patient.

*Amer. Med. Quarterly, June, 1899—Medical Record, New York, July 1st, 1905.

PAPER:

THE CORRECTION OF NASAL DEFORMITIES BY THE USE OF PARAFFIN.

HARMON SMITH, M. D.

So much adverse criticism has been brought to bear upon the subcutaneous injection of paraffin, and so many dangers have been enumerated as possible sequelae to its use, that I have deemed it wise to investigate by personal inquiry the causes which have occasioned the criticism and the conditions which have contributed to the bad results. To this end I sent out to all those engaged in this work a circular letter requesting information as to the following points:

1. Total number of cases injected for the correction of nasal deformity.
2. Number of successful cases.
3. Number of unsuccessful cases.
4. Attributable cause of non-success.
5. Number of cases of blindness due to embolus.
6. Number of cases of sepsis resulting in abscess and loss of paraffin.
7. Number of cases where paraffin lodged in undesirable location at time of operation.
8. Number of cases where paraffin shifted after operation.
9. Instrument used for injection.
10. Kind of paraffin used and melting point.
11. Methods employed to prevent paraffin entering undesirable localities.
12. Conclusions: Do you consider it dangerous, undesirable, unwarrantable, or practical?

I received ninety-one replies to the letters sent. Of this number forty-one surgeons had made paraffin injections for the correction of nasal deformities in one or more cases; forty had had no

experience, and ten had had too little experience to express an opinion. I have not included my own experience in these statistics, but I wish to say that in over two hundred injections for the correction of nasal deformities I have had only two unsatisfactory results, citation of which will be given later.

The history of paraffin injection is now an old story, and I only wish to call attention to the fact that it was first employed for the correction of nasal deformity by Gersuny of Vienna, in 1900.

In the summer of 1901 I began to experiment with the injection of liquid paraffin upon rabbits and upon the cadaver, and in November of 1901 I made my first injection in the nose of a young woman at the Manhattan Eye, Ear and Throat Hospital, at which time I used an ordinary hypodermic syringe and liquid paraffin with a melting point of 110° F. This case is still under my observation, and there is apparently no necessity for further injection. From outward appearances the nose remains the same in size and shape as when it was injected.

Causes of Nasal Deformities—

1. Traumatism. This has been the most frequent cause of nasal deformity in those cases coming under my observation, and I believe it to be the most frequent in the experience of all operators.

2. Syphilis.

3. Abscess of the septum.

4. The removal of the cartilage of the nose in the submucous resection for deflected septa (Wm. E. Casselberry has had such a case.)

5. Lupus of the septum.

Selection of Paraffin and its Preparation. Too much importance cannot be attached to the selection of the proper substance for injection, whether it be purely paraffin or a mixture of paraffin with other substances.

The desirable substance is one with a sufficiently high melting point to reduce to a minimum globular formation when injected, and yet not sufficiently hard to produce inflammatory conditions by constant irritation of the subcutaneous tissues.

Liquid paraffin, or paraffin with a melting point below the body temperature, may readily enter the circulation or gravitate between the loose areolar tissue to undesirable locations, while hard rubber

and celluloid invariably produce inflammatory conditions by their hardness, and slough out.

Emboli have frequently followed the injection of paraffin with a melting point of 92° to 100° F., or when paraffin of a higher melting point has been injected in a semi-liquid state. When the paraffin is liquid or semi-liquid, it permeates the meshes of connective tissue and assumes small spherical shapes, which can unquestionably be taken up by the blood vessels.

My first observations upon the tissues of a rabbit when paraffin had been injected in a liquid state and with a melting point of 110° F. led me to suppose that a fibrosis occurred around each globule of paraffin, but later observations have shown that these globules were only surrounded by connective tissue of the part, and further, that only the larger masses of the paraffin were encapsulated with fibrous tissue when injected cold.

Without citing the various experiments upon the subject, and omitting the arguments of those favoring a soft paraffin, or those advocating a paraffin with a melting point as high as 155° F., I would say that a paraffin with a melting point of 115° F. would meet the requirements, and, if injected cold, would obviate the tendency to embolus formation, and yet be of sufficient hardness to produce a fibrous capsule which would prevent its subsequent shifting.

The conclusions of Luckett and Horn, after repeated experimentation, are that the more solid the paraffin at the time of injection, the more apt it is to be confined to one mass, and the less likely is it to spread throughout the tissues.

In 1904 I removed a section of tissue from the nose of a patient whom I had injected with liquid paraffin two years previously, and the observations made at that time by Dr. Jonathan Wright have in a measure been substantiated by more complete experimentation in the last few years. He called attention to the fact that some of the paraffin must have been carried off by the lymphatics, which was made easier of accomplishment by the fact that the paraffin was injected in liquid form. He further commented upon the specimen, saying: "Had not the experimental observations of others thrown the weight of opinion otherwise, one would naturally suppose from the examination of this specimen that the paraffin on entering the tissues was split up into separate globules, which gradually became surrounded each by its capsule of fibrous tissue. A fluid or semi-fluid, it would seem, would tend to flow along the lymph channels

of least resistance and be broken up into larger and smaller globules. On the other hand, it is a little difficult to understand how the new connective tissue cells can shoot into a non-porous substance. This difficulty of conception is still further increased when we consider the use of melted paraffin which on hardening would seem to offer an impenetrable mass to the invasion of connective tissue cells.

"In the semi-solid compounds with petroleum jelly, such as were used in this case, we, perhaps with less difficulty, can imagine the intrusion of the antennae of an amoeboid cell. Some of the animal

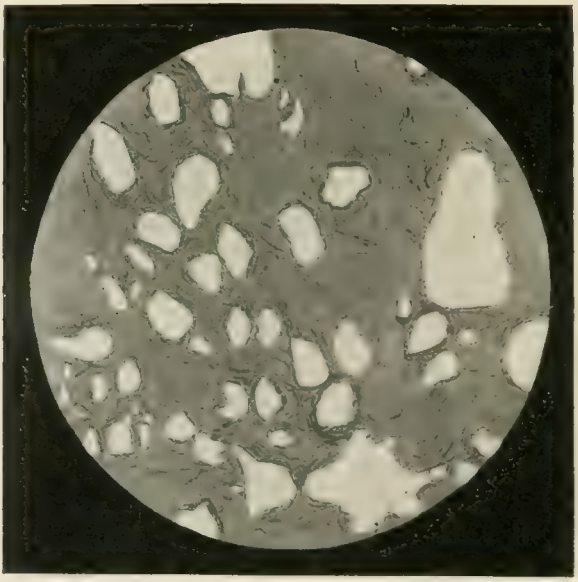


Figure 1. Microscopical appearance of paraffin in the tissues of the nose. Piece removed from nose of patient two years after injection.

experiments are not very satisfactory, when we remember the exceedingly loose subdermal connective tissue, as for instance in the rabbit.

"I would draw your attention to an interesting histological feature shown in Fig. 2, which I think has also been noted by other observers. The latter have been referred to as giant cell formation, and perhaps in their specimens such evidence existed as to warrant the description. I think, however, here it will strike the observer that the large cells imperfectly lining the cavity in which the paraf-

fin lay, are really nature's attempt to line it with the same sort of covering as that which lines the joints, the lungs, etc. Here it is clear these large cells are developed from the endothelium of the lymph spaces. Naturally, with giant cell formation we think of a pathological condition set up by a foreign body—here we think of a physiological, though artificial cavity, a more or less complete adaptation of the organism to new conditions.

"While we should naturally expect in such injections that the paraffin would at first be arranged in one lump, this is not what we should expect in the firmer connective tissue of man. It seems to

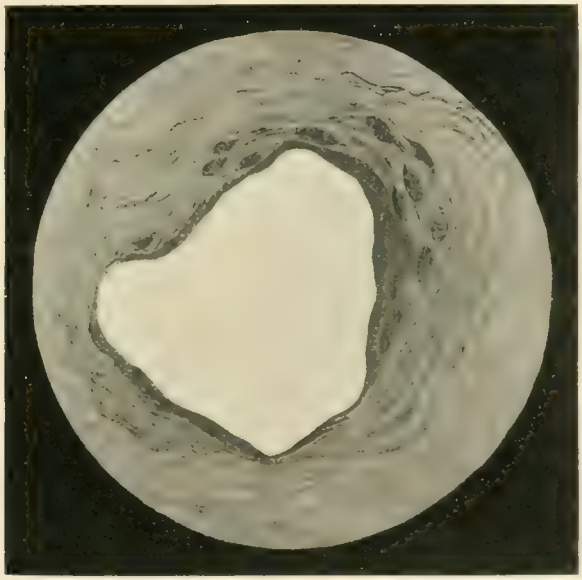


Figure 2. Microscopical appearance of one of the islands of paraffin under high power, showing the endothelial lining of the cavity.

have been shown that this more or less discrete lump in animals, beneath the skin, does finally become split up by the intrusion of connective tissue cells. If this be so, there is no reason why it should not also occur in man, though at first the infiltrating paraffin may be more diffusely scattered through the lymph spaces."

Permanence.—The permanency of the paraffin in the tissues depends upon the melting point of the paraffin and the state at the time of injection. The colder the paraffin at the time of injection,

the more lasting it will be in the tissues, likewise the better the anchorage insured.

Paraffin Mixture.—For a number of years I have used a paraffin with a melting point of 110° F., and the statistics show this to be the melting point most used. This paraffin was obtained by adding to commercial paraffin melting at 140° F. sufficient petroleum jelly to bring it down to 110° F. However satisfactory this paraffin has proven in my hands and in the hands of my colleagues, I am now inclined to believe that a higher melting point would be more practical, and have recently used a paraffin prepared in the same manner, but with a melting point of 115° F. This, if injected cold, will be even less liable to assume a small globular shape, and will reduce still more the danger of an embolus. It has also the advantage of forming for itself a berth of connective tissue which will insure against subsequent displacement. A third point in its favor is that in very hot climates, or when a patient is subjected to extreme heat, there will be less tendency to soften.

The liquid paraffin called albolene is decidedly preferable to vaseline in reducing the melting point of the hard paraffin. Various combinations have been made in the preparation of a suitable substance for injection. White wax, rubber and wax, and rubber and paraffin, all have had their advocates. However, I can find no possible objection to the hard and soft paraffin, properly prepared, and with a melting point of 115° F.

Instruments for Injection.—At first it was deemed necessary to keep the paraffin liquid in order to inject it, and to meet this requirement numerous ingenious devices were constructed. Quinlan devised a syringe with a hot water jacket which was supplied constantly from a tank of hot water; Eckstein placed rubber tubing over the barrel of his syringe to preserve the heat; Walker Downie constructed one with the outside surrounded with a continuous platinum wire, which was kept hot by a current of electricity. All of these syringes having the preservation or the generation of heat as their object, were readily supplanted by one capable of expelling the paraffin in a solid state.

I devised a metal syringe with a screw piston, which would enable the operator to expel the paraffin cold. This syringe was presented to the American Laryngological, Rhinological and Otological Society in Washington in May, 1902, and was the first of its kind, so far as I know. This syringe possesses all of the qualifica-

tions necessary for the injection of solid paraffin, and has the advantage over a ratchet injector in being able to control the amount of injected paraffin to a fraction of a drop; while the injector necessitates injecting all of that which is included in the space controlled by a notch of the ratchet. (Figure 3, paraffin syringe.)

Broeckaert was the first to introduce the ratchet injector, since which others have added to the number. The injector has the advantage over the syringe in that one may inject with one hand and mould the paraffin with the other. It is also a better instrument for injecting turbinates, in cases of atrophic rhinitis. Pfau has manufactured a very complete instrument of the injector variety.

However, for practical purposes my screw syringe is as safe and satisfactory as any instrument that I know of. I believe a curved

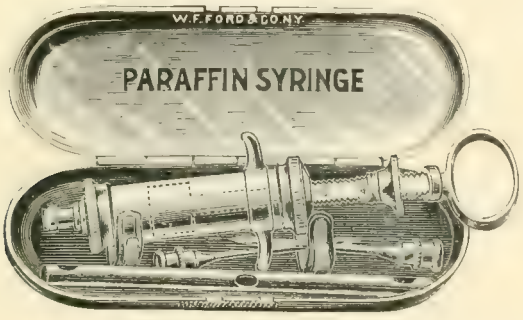


Figure 3. Paraffin syringe

steel needle of large calibre and two inches long is the one best adapted to the purpose.

Suitable Cases for Injection.—Too much stress cannot be placed upon the selection of suitable cases for injection. The majority of bad results may be attributed to the improper selection of cases for operation. Syphilitic cases in which some activity is still present should not be injected, nor should any specific case be operated upon until a thorough anti-syphilitic treatment has been administered for some months. No case should be injected where all of the cartilaginous septum has been destroyed, as there is nothing below to support the paraffin above. No diabetic nor nephritic case should be injected, as the low vitality of the tissues will not stand an increased strain. Scar tissue is always poorly nourished, and great care and judgment should be exercised in the attempt to liberate it from its source of nourishment and to make upon it an

additional call for self-maintenance. Small quantities of paraffin should be injected beneath scar tissues at long intervals. No attempt should be made to inject beneath tightly adherent skin where an additional tension will occasion pressure, for in such cases necrosis is likely to occur.

Furthermore, I do not believe in injecting any case unless deformity exists which detracts materialy from the appearance of the patient and renders him peculiarly sensitive to his appearance. The injection of paraffin to overcome slight inequalities of profile and to gratify the whim of some over-sensitive patient is unwarrantable, in view of the fact that quite a number of cases have proven unsuccessful in their ultimate outcome.

Preparation of the Patient.

1. Scrub the nose and adjacent areas with green soap and water.

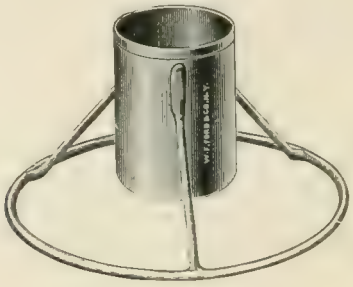


Figure 4. Paraffin cup.

2. Pour on some alcohol.

3. Cover the head with a towel wet with bichloride of mercury, and cover the arms and shoulders with a sterilized gown. The same preparation should be made here as for any minor surgical operation.

Preparation of Instruments and Operator.—The paraffin syringe, which is of metal, and the needle should be boiled. The paraffin, which comes in sterilized tubes, should be again boiled in a metal cup, which can be placed in any sterilizer, the bottom of the cup being raised sufficiently to prevent the paraffin from scorching. The paraffin is drawn up into the syringe in a liquid state, after which the syringe is dropped into a receptacle of cold sterilized water, which soon solidifies it. The hands of the operator and the assistant are both sterilized. (Figure 4, paraffin cup.)

Methods of Injection.—No anesthetic is necessary, although some operators prefer cocaine locally injected. I have found the cocaine as painful as the paraffin injection. I have the patient sit on a stool so that when the head is tilted backward the nose is about the height of the operator's elbow, with the arms hanging by the side. The operator stands on the left of the patient, and the assistant stands in front of and slightly to the right of the patient. The assistant grasps the nose firmly with the balls of his thumbs pressed against the nasal bones, and with the tips touching over the root of the nose. In this way pressure is exerted along both sides of the nose, and will prevent the entrance of the paraffin into the areolar tissue around the eye, and will also prevent it getting into the circulation, should the needle penetrate a small vein. I always inject from above downward, as this is in a direction away from danger, and toward nature's natural barrier -which is the adherence of the skin and cartilage of the tip and alae of the nose.

Before the introduction of the needle I immerse it in hot water, and then give the piston several turns until the paraffin comes out in a hard cylindrical thread. The first few turns of the piston usually sends forth an interrupted stream of paraffin mixed with some oil and water, but in a few turns all of the oil and water have been expelled, and the paraffin remains, a solid block within the cylinder of the syringe.

At the point of injection I lift the skin up high with firm pressure and introduce the needle beneath the skin and into the areolar tissue above the periosteum. The point of the needle is carried just beyond the depression, when the injection is begun slowly and is continued as the needle is gradually withdrawn. I frequently stop the injection and mould the paraffin to meet the requirements of the case. During this time a second assistant holds the syringe, or the first assistant relinquishes his pressure and holds it while I note the results. I never make it an object to overcome the deformity at one injection, but in many instances I have found only one injection necessary. When anemia of the surface occurs I invariably stop further injection, as this is the danger signal that the tissue will stand no more. I withdraw the needle carefully and stop the hemorrhage, if there is any, with adrenalin, after which I put on a little collodion. If convenient for the patient to do so, I advise rest in bed for the remainder of the day, and the application of ice cloths. When the paraffin melts at 115° F. and is injected

cold, it enters the tissue as a hard mass and cooling sprays are unnecessary.

A second injection should not be made sooner than a month afterward, for nature is having all she can do to take care of this first injection during this time, and any additional demand might result in necrosis.

Accidents.—The discussion under this head will be limited to those accidents resulting from injecting paraffin for the correction of nasal deformities.

Embolus.—There are three authenticated cases of thrombosis of the eye vessels immediately following the injection of paraffin for nasal deformity:

1. Amaurosis of the left eye, caused by thrombosis of the opthalmic vein, reported by Leiser in 1903 (Berliner Klinische Wochenschrift, 1903, p. 13.)

2. Amaurosis of the right eye, caused by thrombosis of the arteria centralis retinae, reported by Hurd and Holden in 1903. (Medical Record, July 11, 1903.)

3. Amaurosis of the left eye, with temporary hemiplegia of a mild character which disappeared in a few days, reported by Thos. H. Huntington, in November, 1904.

The report of these three cases immediately prejudiced nearly every operator against the use of paraffin, without a consideration of the probable errors entering into the operation. These errors I believe are absolutely preventable. Should all hypodermic injections be given carelessly, without expelling the air bubble, I have no doubt the death rate from air embolus would increase amazingly, yet no physician hesitates to give a hypodermic. Here is a possible error easily avoided. In all three cases referred to above the accident occurred at the time of a second or third injection. The fact that thrombosis occurred in the second and third injection in each case would tend to show that the increased vascularity of the area favored the transmission of any small particle of paraffin away from that location, whether it be a part of the newly injected substance or a detached portion of that previously injected.

In case II the paraffin was injected in a semi-solid state; and in case III it must have been in a liquid state, as Dr. Huntington tells me by letter that he used an ordinary hypodermic syringe, by which only liquid paraffin can be injected. In case I the method

of injection is not given, although the melting point of the paraffin is given at 110° F.

In none of these cases is any mention made of protection, by pressure, to the soft tissues around the eye, against the invasion of the injected substance. Hurd's case was the only one where the injection was made from above downward, and this was immediately following one made from the tip upward. I believe these accidents were due *primarily* to the absence of protection by pressure at the sides and root of the nose against the migration of paraffin particles, and *secondly* to the use of liquid or semi-solid paraffin. Had a solid paraffin been injected, with a melting point of 110° F. or above, and the injection been made from above downward, and firm pressure been made at the root of the nose, as I have indicated, I believe the accidents might have been avoided. I have reports by letter of 1,250 cases of paraffin injection, in which number three cases of thrombosis have occurred. In ordinary mercury injections there are records showing as high a proportion as 8.9 per cent of embolism (Luckett and Horn.)

Four conditions favor abscess formation:

1. Unsurgical methods, resulting in infection.

2. A second injection following too closely upon the first. This error resulted in one of the unfortunate experiences I have had in the injection of paraffin.

The case was sent to me by Dr. L. G. Woodson, of Birmingham, Ala. The tissues were very tight over the depression, and at the time I advised against an endeavor to overcome all the deformity in one week, the time the patient had arranged for his stay in New York. However, I made two injections in the seven days, and the patient returned home perfectly satisfied with the result. I should have considered the operation a success had I not interviewed Dr. Woodson some months later, at which time he told me that in about two weeks after the patient returned home the tissues broke down and most of the paraffin exuded, although sufficient of it remained, or sufficient fibrosis resulted from the resulting inflammation to completely overcome the deformity. I had added here additional work to an already overworked area, with the result that the vitality of the tissue was overcome. A similar instance is reported by Richards, of Fall River.

3. Abscess with sloughing occurs in diabetic, nephritic, and syphilitic cases.

4. Infection and sloughing occurs when paraffin is injected into the skin instead of beneath it. The skin is poorly nourished over these deformities, and injection into its layers still further reduces the blood supply to that particular area. Again, infection may readily travel through a sweat gland if the injection is made too close to the surface. (Richard Lake, of London, reports one such case.)

It would certainly reflect upon one's surgical judgment to offer confident solutions of all these problems, but the mere statement of how they occur suggests the method by which they may be obviated.

Distribution to Undesirable Locations.—All of the reported cases where paraffin has entered undesirable localities not intended for injection have been when the injection was made with a semi-liquid substance, with the needle pointing upward, and with insufficient precaution in regard to pressure.

With a solid paraffin, a screw syringe, with the needle pointing toward the tip, and with the fingers of an intelligent assistant to aid in the procedure, I believe there will be no case where paraffin will lodge in any other locality than the one desired. There may be one exception to this statement, and that is where considerable scar tissue exists at the site of the deformity, and the precaution to elevate this with a bistoury is not properly accomplished before the operation. Then, of course, the paraffin would go in the direction of least resistance.

Shifting of Paraffin Subsequent to Operation.—There are seven cases reported in my 1,250 cases operated upon. To this number I can add another of my own, which I have not seen, but which has been reported to me by Dr. W. E. Casselberry of Chicago. In this case I am not sure that the paraffin shifted, as I am of the opinion that I injected a column of paraffin along the ala of the nose to see if it would not aid in overcoming a relaxed ala. Of course paraffin may shift, but it has been clearly demonstrated by experiment upon animals that hard paraffin will create a fibrosis around the mass, while a soft paraffin will gravitate through the areolar tissue. To lessen the danger of shifting, inject the paraffin cold, as the nearer it approaches a discrete mass the more readily will a fibrosis form around it.

Keloid.—E. Fletcher Ingals of Chicago writes me that he considers the injection of paraffin dangerous owing to the danger of

death, and to the fact that there have been at least eight cases of keloid following its injection. There is no doubt that a fibrosis is set up by paraffin injections, varying in intensity with the hardness of the paraffin. A paraffin melting at 155° F. would tend to produce as much fibrosis as a plate of hard rubber or celluloid. Keloid has never occurred where lower melting points have been used, so far as I know. I can see no reason why keloid should form when a paraffin of 115° F. melting point is used. I have never seen a case, and have never heard of a case occurring in the experience of any of my co-workers. Dr. Ingals tells me that these cases did not come under his personal observation.

Carcinoma.—Dr. R. Bishop Canfield of Ann Arbor, Mich., has given me by letter the only report of a case of this nature in any way connected with paraffin injection. The case is of sufficient interest for me to give it in full:

"In connection with your paper you will be interested in the following case which presented itself at my clinic some weeks ago. A young boy, 17 years old, in whom nothing suggestive of syphilis could be discovered, with a history of having had a marked deformity of the nose corrected with paraffin some months before, showed a nose about twice the size of the normal, rounded on all sides and almost shapeless, red in color and giving the impression when palpated of being very dense. In both very hot and very cold weather it was bright red with dusky blue red spots. The patient said he knew that paraffin melting at 110° F. had been used, and that the nose was somewhat larger and redder now than it was some two or three weeks after the injections. It was so mortifying to the patient and was so dense that I determined to try to dissect some of the paraffin out. I neglected to say that the entire septum was about half an inch thick, and apparently filled with paraffin. I made a free incision into the skin over the area of greatest density, first on one side and then on the other, in the hope that I might be able to express out a little of the paraffin, but was unable to do so. The tissue *bled* very freely from all the cut surfaces. I removed pieces for microscopical examination. After the incisions healed there was, to my surprise, considerable improvement in color, which was decidedly less red than before, due, no doubt, to decrease in pressure following removal of the pieces of tissue. Microscopical examination by Dr. Warthin shows the perfect picture of squamous carcinoma. I have not seen the patient

since a week after the operation. He has promised to return if the nose increases in size. If he does, I will let you know how he is."

In view of the fact that this patient's septum was filled with paraffin, it is reasonable to suppose that some growth existed before the paraffin was injected. I believe the new growth in this case was a coincident, and that the cancer was not the result of paraffin, but was possibly the cause of the condition for which the paraffin was used.

Figure 5. Side view, showing marked depression. Before injecting paraffin.

Figure 6. Front view. Before injection.

Advantages Over Other Surgical Measures.—Paraffin injected under proper surgical conditions and with the precautions advised in the preceding paragraphs will remain in the tissues and serve its purpose far better than the surgical introduction of silver, aluminum, or hard rubber plates, which invariably slough out. In the correction of nasal deformity by breaking up the nasal bones and breaking away the nasal processes of the superior maxilla to aid in forming the bridge, there is ordinarily too little septum to give the necessary support to insure a permanent structure, and the seriousness

of this procedure, with the necessary sojourn of the patient in the hospital, is a question which frequently determines the patient against the operation. Neither a general nor a local anesthetic is necessary in the injection of paraffin, and the patient may continue his work without scar or discomfort.

The history of the following case demonstrates the superiority of paraffin injections over the surgical insertion of silver or celluloid plates for the restoration of nasal deformities.

The patient, Mrs. N., was referred to me by Dr. George E. Brewer, August 7, 1907. She was 37 years old, and when a child her

Figure 7. After injection.

Figure 8. After injection.

nose was broken. An abscess of the septum followed, which resulted in loss of the septum and a saddle-back nose. Fourteen years ago she consulted Dr. W. T. Bull, who inserted a silver plate for the restoration of the bridge. This plate remained in situ for three years and gave satisfaction. At the end of this period inflammatory conditions began, which soon resulted in the extrusion of the plate. The patient then consulted Dr. Robert Weir, who inserted a celluloid plate, which came out in six weeks. Examination on entrance to the Manhattan Eye, Ear and Throat

Hospital showed complete loss of all the cartilaginous septum, nearly complete atresia of both vestibules, and marked saddle-back nose, with adhesions and cicatrices binding the skin close to the bones. My attention was directed first to the atresia, which was satisfactorily overcome by January 2, 1908, after the removal of nearly all the scar tissue in each vestibule, and the insertion of hollow splints for five months. I made the first injection of paraffin on the above date, and a subsequent injection two weeks later. The first injection was followed by no reaction whatever, but the second was followed by some edema under the eyes, and redness and swelling over the nose—all of which finally subsided, and the result was obtained as seen in the photographs (figures 7 and 8).

Deductions from Statistics.—

Total number of cases reported by forty-one different observers, not including my own—1,252.

Of this number:

1,000 were entirely successful.

104 were unsatisfactory.

2 of the 104 cases resulted in blindness.

55 of the 104 cases resulted in sepsis and extrusion of the paraffin.

In 22 of the 104 cases the paraffin lodged in the wrong location.

In 7 of the 104 cases the paraffin shifted after operation.*

The instruments used were ordinary hypodermic syringes, Killian's, Beck's, Pfau's, Broeckaert's and Smith's. The paraffin injected varied in melting point from 103° to 136° F., the majority using a paraffin melting at 110° F., and with the proportions originally suggested by the writer. The precautions against the distribution of the paraffin in undesirable localities were digital pressure and plates of lead and steel.

Thirty-one of the forty-one operators consider the procedure practical if proper precautions are observed, and if performed by an experienced operator. The other ten consider it dangerous, and in most cases unwarrantable. I have tabulated, as far as I could, the replies to my circular letter, and submit the result to you for closer scrutiny. I have given the names of those signing the letter, as I presume they gave consent to using their names by

*These figures will not sum up correctly, as some of the operators gave only their number of unsuccessful cases and omitted their total number of cases operated upon.

so doing. Those who did not sign may recognize their own statistics with their names left out.

Conclusions.—This operation is not without its dangers, which, however, now that we know of them, can be avoided. It is a practical procedure when performed by one of experience and with thorough surgical precautions.

The proper paraffin is one with a melting point of about 115° F. and made of hard and soft paraffin. The mixture must be injected cold, and the needle directed from above downward. The upper part of the nose must be protected by the digital pressure of an assistant, and the injection must be made slowly, and only in small quantities at each sitting. The second injection should not be made shortly after the first, but ample time should be given the tissues to regain their normal vitality before injecting more material.

Cold compresses after the injection favor the anchorage of the paraffin, and a collodion dressing over the entrance of the needle is a precaution against infection.

No syphilitic, diabetic, nor nephritic patient should be injected without due consideration to what has already been said; and, lastly, no attempt should be made to inject cold paraffin except with a screw syringe or with one of the ratchet injectors.

PAPER:

THE REPAIR OF HARE-LIP AND CLEFT PALATE.

J. F. BARNHILL, M. D.

The repair of hare-lip and cleft palate has heretofore been largely left to dentists and the general surgeon, chiefly, no doubt, for the reason that, as laryngology and rhinology developed to its present state in a comparatively short time, those who practiced these specialties have been kept sufficiently busy in perfecting operative procedures which have seemed more intimately connected with these fields. Moreover, only recently the specialist in diseases of the upper air tract was presumed to be more of a therapeutist than a surgeon, his training was usually not that of a surgeon, and hence, he was not often capable of successfully carrying out the complete and often difficult details of the surgical steps necessary to the closure of these congenital defects.

At the present time, however, when many of the problems connected with both the surgical and medical aspects of the diseases of the upper respiratory tract are more or less settled questions, and when many who limit their practice to these diseases have found that they must, of necessity, be surgeons, and have accordingly received ample training in the principles and art of modern surgery, the occasion seems now at hand for the laryngologist and rhinologist to claim his own, and to undertake this class of work, which, in the main, is less difficult than much of that which he is already doing. The repair of hare-lip and cleft palate should be included in the practice of laryngology and rhinology for the following reasons: 1st. The defects involve the structures of the upper air tract, often converting the mouth, nose and naso-pharynx into one cavity. 2nd. Voice production, or, at least, normal speech, is always greatly impaired. 3rd. The repair of the clefts involves the restoration of the nose and soft and hard palate to as nearly the normal as possible and in doing this the muscles of

the soft palate and those which to a great extent regulate the patency of the Eustachian tube, are involved in the surgical repair. 4th. No one is presumed to have a better knowledge of all these structures and their functions than the laryngologist, and no one should be better able than he who is thoroughly trained to work in such cavities to carry out the delicate technic necessary to success.

Statistics concerning the frequency of hare-lip and cleft palate vary somewhat, but an average seems as follows: One cleft palate in about every 5,000, and one hare-lip in about every 2,500 infants. Hence these affections are the most common of the congenital defects of the lip and mouth. The cause is a failure of union on the part of the several embryonic processes necessary to perfect development. The reasons for such failure to unite assigned by various writers are heredity, maternal impressions, and maternal disease and malnutrition. Arrest in the embryonic development of the embryonic structures concerned is often assigned as the cause of the cleft, but Brophy maintains that the parts are fully developed and that the cleft is solely the result of their failure to properly coalesce.

Time for Operation. Most surgeons now agree with Lane, Brophy and Ferguson that the best time to operate is soon after the birth of the child, many preferring to do so, especially when the defect is only a hare-lip, on the day following birth. Of course, should the infant be badly nourished, or in some other way affected, it would bear any operation badly and the procedure should be delayed until the causes calling for the delay have been remedied. If at birth the child is plump and well in every way, except the hare-lip or hare-lip and cleft palate, operation at this time is best, for if postponed the child will probably be found in a less satisfactory condition very soon for the reason that its defects prevent in a marked way its proper nourishment. Should the child have approached the age of dentition before consultation with the surgeon, it is wise to allow the teeth to erupt before the procedure for closing the gaps is undertaken, although infancy is the proper time to operate for the reason that at this age not only can the gaps be properly closed, but by doing so at this time the chances in favor of better phonation and satisfactory speech are greatly favored. Nevertheless, should operation be refused or neglected in early life, and the patient be seen in youth or adolescence, good cosmetic effects are often obtained, even though but little improvement

in speech is possible and operation should not, therefore, be refused at any age.

The operations for hare-lip and cleft palate can be best described separately.

I. Hare-lip.

This defect may be single or double, and may or may not be complicated by cleft palate. The lip defect may amount to only a small notch, which is scarcely noticeable, or it may extend into the nostril, and involve the alveolar process and superior maxilla toward and rarely even to the orbit. When the defect is double, the intermaxillary portion of the alveolus covered by rudimentary portion of lip projects forward at a varying angle to the

Figure 1. Child age one year. Double hare-lip with projecting intermaxillary bone.

natural plane of the alveolar arch, to which in some cases it is attached at almost a right angle to the tip of the nose, and anterior septal margin.

The plan of operation in any case must always depend upon the nature and extent of the defect. Each case should therefore be carefully considered upon its own necessities. In case an incomplete notch only is present, the operation for its correction is simple, and Nelaton's method of procedure can be successfully followed. This consists in transfixing the lip parallel to the mucocutaneous border of the notch for a distance dependent upon the depth of the defect, and at a distance from this border of from one-quarter to one-third of an inch. The lower portion of the

incision is then pulled downward into a normal position when retention sutures are inserted.

When a single fissure extends up to, or into the nostril, either a unilateral or bilateral tongue-shaped flap may be cut from each lip, the plan to be followed depending upon the amount of tissue which is missing. The question of the number of flaps, their width and length, is a question in all cases to be settled by the operator at the time of operation. The principle should always be followed of utilizing all the tissues that remain, to the end that as little subsequent tension as possible will be exerted on the retention sutures. In this respect Ferguson's plan of turning back flaps of mucous

Figure 2 Same as figure 1 three months later, and after operation.

membrane from the upper three-fourths of the cleft, first uniting these, and then suturing the lip-margins is most excellent.

In cases of double hare-lip with intermaxillary projection which stands at right angles to the normal alveolus, and which is adherent to the tip of the nose, it is necessary to deal with such projection by a preliminary operation in which the misplaced part is dissected loose from the nose, and its osseous junction with the superior maxilla is fractured to such an extent as to permit its replacement in a position corresponding with the normal alveolar arch. In addition to this it is best to freshen the adjacent edges of both the replaced intermaxillary process and the alveolar arch, and to approximate the same by means of silver wire sutures. Two weeks or more may then be allowed before the hare-lip is repaired.

I know of no condition that would justify the complete removal of the intermaxillary portion, although this is advocated in several standard works on surgery. In case the deficiency extends into one or both nostrils, and as a result the cartilaginous wings are distorted and detached from the septum, the plastic repair must include the careful restoration of the same by their approximation and retention to their normal relation to the nasal septum. Some-

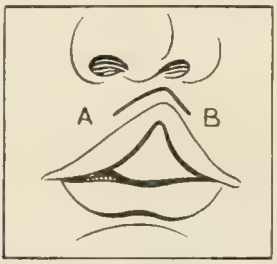


Figure 3. Nelaton operation. AB, line of incision through lip.

Figure 4. Nelaton operation; lip depressed. Line AB thrown vertical.

times this requires the resection of a portion of the cartilage in order to secure a proper contour of the nostril. In shaping the nostril much assistance may be had by using a sound or bougie, over which each nostril wing is moulded and secured.

The failures resulting from repair of hare-lip are due, 1st, to malnutrition of the patient; 2nd, to improperly prepared or bad-

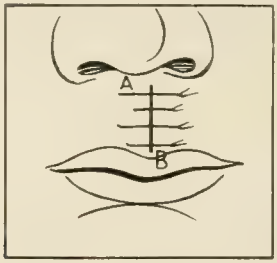


Figure 5. Nelaton operation, suture inserted.

ly adjusted flaps, and 3rd, to too great tension on the sutures resulting from facial movements, or to the fact that the sutured portions of the lip were drawn into position without sufficiently releasing the attachment of the lip to the alveolar processes. The first and second of the above reasons for failure always involve the judgment of the operator, and do not, therefore, need discussion here. In every operation for hare-lip the amount

of tension necessary to bring the separated portions of the lip together should always be tested, while the patient's face is in absolute repose, and unless the parts fall together quite readily and naturally, a small knife or a straight, sharp scissors should be thrust through the mucous membrane of the lip at its junction with the alveolus and the labio-alveolar separation accomplished through the smallest possible incision. Facial movements may be controlled,

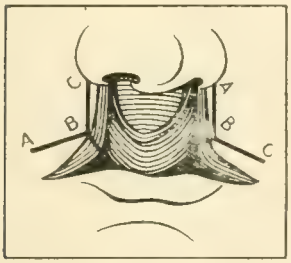


Figure 6. Operation for double hare-lip: ABC, lines of incision for making flap.

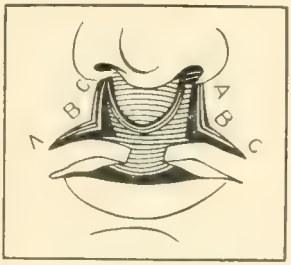


Figure 7. Double hare-lip. Construction of flaps.

and the operated part put at complete rest by carrying a strip of adhesive plaster three-quarters of an inch wide over the line of incision from one cheek to the other. Since this often becomes quickly soiled from nasal and mouth discharges, and requires fre-

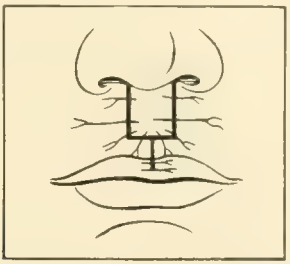


Figure 8. Double hare-lip. Flaps, adjuster and sutures in place.

quent changing, much annoyance and pain are produced by necessary changes. An appliance which I have seen used successfully, and which I have employed myself with satisfaction, is that of H. R. Allen, which is made from a watch spring, and sets upon feet which are broad enough to distribute the pressure from the spring over a sufficient area on each side to entirely relieve all tension upon the sutures. A chief value of the device is that dressings may

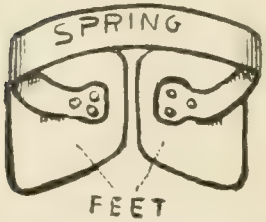
be made for any necessary length of time without the slightest necessity of changing the instrument. (See figures 9 and 10.)

II. Cleft Palate.

Operative measures directed to the closure of cleft palate are more severe than those for the repair of hare-lip. The difficulties attending the performance of the former are greatly multiplied in comparison to the latter. Surgical anatomy, the mechanics of surgery and a thoroughly known surgical technic are necessary acquirements on the part of the surgeon.

Choice of Time for Operation.

It was formerly thought advisable not to operate for cleft palate before the age of two years.* Most operators at the present time



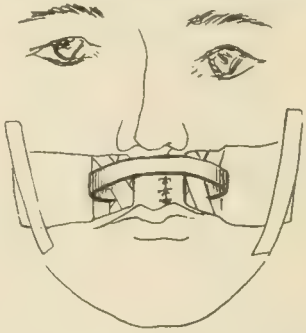


Figure 9. Allen's hare-lip spring. The feet are pulled apart before applying to the operated lip. The pressure of the spring holds the sutured parts snugly together till healing takes place.

Figure 10. Allen's hare-lip spring applied, showing adhesive plaster fastenings. Antiseptic gauzes are readily applied between the lip and spring, and these can be changed as often as necessary without in any way disturbing the wound.

prefer operation during early infancy. Brophy, whose experience entitles him to speak with authority, states that the best age to select is within three months from birth, and he frequently closes the defect as early as the tenth day.

Briefly stated, his reasons for early closure are as follows: 1. The surgical shock is less. 2. The bones of the face at birth are pliable and malpositions may then be more easily corrected. 3. The earlier the correction of the defect, and therefore the earlier that the mucous and muscular structures are put into their new and more normal positions, the better will be the results as to voice and

*Augenbach and Trelat preferred operating at seven years. Roux believed operation should be delayed as late as sixteen years. Kirmisson, of Paris, does not state an exact age of choice, but indicates that it should not be done during the early years of childhood.

speech. 4. A more normal shape and position of the superior maxillae, and especially of the alveolar process is established by early closure.

Technic of the Operation.

This depends greatly on the age of the patient at the time the closure is undertaken. When the child is seen at birth, or during the first three months following, I believe the plan of procedure advocated by Brophy is best. This operator has shown to my own satisfaction that the cleft in the palate is not due, as usually stated,

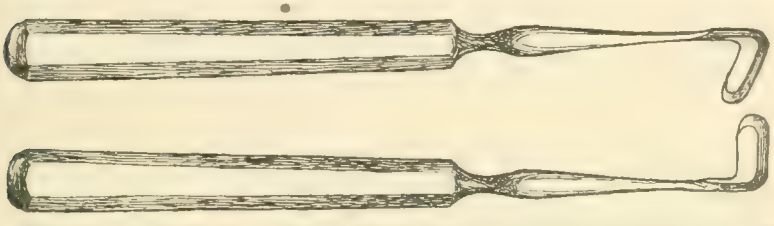


Figure 11. Brophy's curved periosteotomes used in the elevation of the soft tissue of the hard palate. There are a number of these instruments varying from a right angle to the acute angle shown in the drawing.

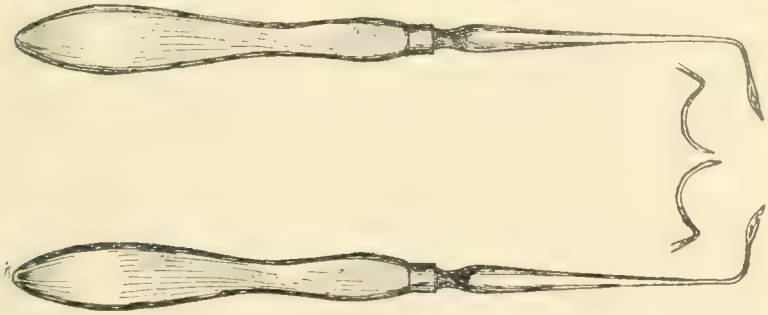


Figure 12. Deschamp's needles used in the introduction of tension sutures.

to an *arrest* in development, and a consequent *deficiency* of tissues, but that the fissure occurs as the sole result of the failure of the two sides *to unite*, even though sufficient material is present. The failure, therefore, being one of lack of union, the logical procedure would seem to be one of bringing the two sides of the head into apposition and of anchoring them there until union takes place. This Brophy accomplishes by the following steps:

The infant is prepared by cleansing the mouth with antiseptic washes. Chloroform is administered through the tube of a special device. A mouth gag is inserted. (See Figs. 11, 12 and 13 for the several instruments necessary to this operation.) The edges of

the cleft are denuded, including a shaving from the bone, if thought necessary. The cheek of one side is then retracted, and a specially devised needle which is threaded with braided silk is inserted into the alveolar process from the outside, at a point near the posterior margin of the hard palate, and on a level with the horizontal table of the palate bone. It is thence pushed inwardly through the substance of this plate until the point emerges in the central cleft and the silk braid is released. This same procedure is repeated on the opposite side. By attaching one of the silk threads to the other in the cleft, a single thread is drawn from the exterior of the alveolus on one side, through the intervening bone, across the cleft, to the opposite side. A No. 20 silver wire is then attached to the silk thread and drawn through to the opposite side, thus substitut-

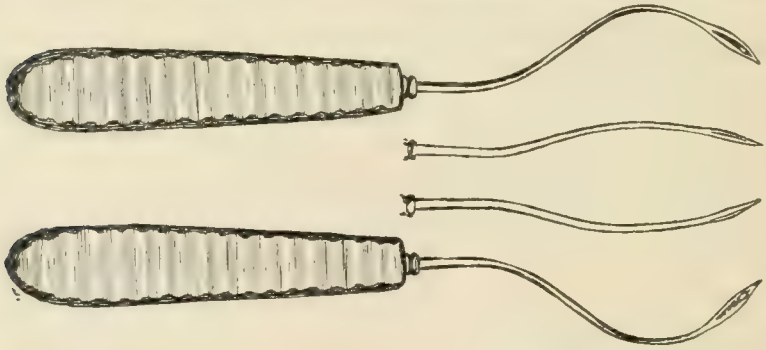


Figure 13. Brophy's strong needles used in the introduction of silver tension sutures through the maxillary bones.

ing the silver for the silk sutures. Near the front of the alveolus a second wire is passed from side to side in an exactly similar manner as the first, only that the wire is carried above, and not through, the bony structure of the hard palate.

In order to prevent these silver sutures from cutting out, two lead plates, one for each side of the alveolar process, are shaped to fit the respective processes they are to cover, are perforated by two eyelets which are the same distance apart as are the two wires at the point of emergence, and through these the wires are passed, drawn tightly, and twisted one to the other. The lead plates are well tolerated, and effectively prevent the wires cutting through the alveoli. Force applied by means of a specially devised clamp may be necessary to approximate the two lateral halves of the jaw during the twisting of the wire sutures.

In case there is a wide gap between the margins of the palate bones and the structures to be approximated are unduly resisting to the tension of the wires, it may be necessary to cut through each malar process. If this be done, the bone should be severed through a small submucous incision, since otherwise the fractured alveolus would be inefficiently supported, and might be readily displaced into a malposition.

The line of approximation of the two mucous edges of the cleft should be examined on the oral surface, and should they not lie in perfect apposition any required number of fine silk sutures may be inserted before considering the operation finished.

In operations performed on infants having both hare lip and cleft palate, the palate should be repaired first, for the reason that the extra space given to the mouth on account of the lip defect aids greatly in carrying out the necessary steps of the palate operation.

Operation on Cases More Than Three Months Old.

In the event that the individual has passed the age when it is longer possible to approximate the two lateral halves of the superior maxilla, as just described, some form of flap closure is usually to be chosen, and from the many plans advocated by various operators I have employed the one known as the Davies-Colley operation. One of the chief difficulties in any plan of procedure is to overcome the tension upon the sutures during the time required for union of the fissured structures, and the method suggested by this surgeon overcomes this as well or better than any other I have tried.

The operation consists in the dissection, from the palatal surfaces, of three flaps, and of making one incision. It is sometimes called the double-door operation, for the reason that two of the flaps swing together on hinges from the whole distance of the marginal clefts. These flaps are so turned and permanently fixed during the operation that the upper surface of the whole cleft is covered by mucous membrane taken from the oral surface of the palate, and the oral surface of the cleft is reinforced and greatly strengthened by a large muco-periosteal flap which is turned across the cleft with its mucous surface looking downward. This arrangement brings the periosteal surfaces of all the flaps into direct apposition with each other, and in these osteoblasts form, and a more nearly normal palate may be expected. Moreover, the pressure of the patient's tongue against the roof of the mouth,

which will be more or less firm after any operation, will, in this one only, press the two raw surfaces more firmly together, and will, therefore, favor rather than hinder the reparative process.

Descriptions of operations like this are worthless and I therefore refer to the figures. The steps are, 1st, an incision A B close to the alveolus, and with its center at the last molar tooth. The incision is made down to the bone over the hard palate, and transfixes the soft palate for a distance dependent somewhat upon the width of the cleft. The tensor palati and levator palati muscles of one side are, of course, divided by this incision. An incision is then started at C, and is carried downward on the same side about one-quarter on an inch from the border of the cleft through the

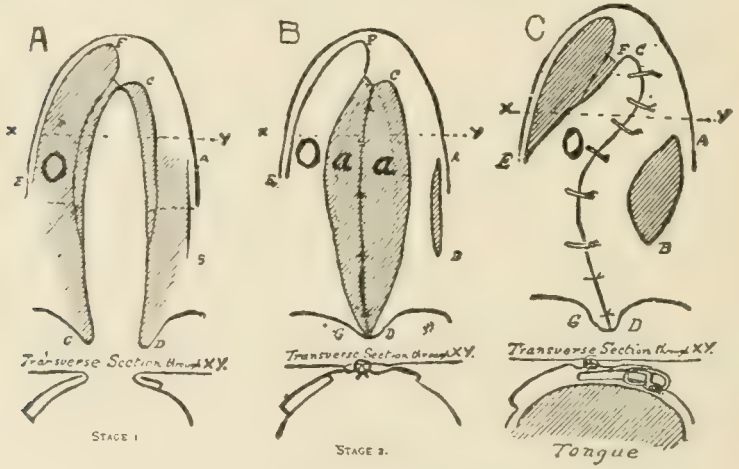


Figure 14. Davies-Colley Operation.

length of the hard palate, but as the posterior border of the hard palate is reached, the incision is brought to the margin of the cleft, whence it is continued in such way as to simply divide the two surfaces of the soft palate and uvula, the incision finally ending at D. Another incision is begun at F on the opposite side and continued along the border of the cleft of that side at a distance of one-sixth to one-fourth inch, until the posterior edge of the hard palate is reached, at which point the cut is brought to the edge of the cleft, and continues as a splitting incision of the anterior and posterior mucous surfaces ending at G. A fourth incision is begun at E and continued to F, thus completing the outline for the third muco-periosteal flap.

2nd. The next step of the operation consists in the elevation of all these flaps. By means of one or more elevators of suitable size and curve, the flaps are loosened from the bone over all the shaded areas shown in Fig. 14, A.

3rd. The two marginal flaps are then turned toward each other and sutured as shown in Fig 14, B. This accomplished, flap O is turned across the field of the sutured flaps a a, and is sutured into the position shown in Fig. 14, C. In order to relieve any undue tension that may be found present at the time of introducing the sutures, it may be found necessary to lengthen incisions A B, or to carry the incision at E to a lower point, and to elevate the muco-periosteum over a greater area. The suture material may be ten day chromic catgut, silk or wire, according to the preference of the operator. Davies-Colley recommends fine silver wire.

The after treatment is much the same following any plan of operation. The mouth and line of sutures should be gently sprayed with normal salt solution several times a day. The nourishment should receive very great care. The child should be given its milk from a spoon, and not from the nipple of a nursing bottle, which would likely disturb the sutures. The child should be kept in the open air and sunshine if the weather will permit, and if not, the ventilation should be the best possible to secure. Provision must be made for preventing the infant cramming its fingers or hands into its mouth.

Results.

If the operation for hare-lip and cleft palate prove entirely successful two benefits should be noted, viz.: The marked change of feature and an improvement in voice and speech. The former is, of course, immediate, while the degree of benefit to the voice and speech can only be estimated as the child acquires a knowledge of spoken words. As a rule, the earlier the operation for cleft palate is performed, and the smaller the amount of injury done to the palatal muscles during the operation, the better will the voice be. Following operations in adult life, especially if the palatal muscles have been severed, and much thickening and scar tissue of the soft palate results, very little good will result to the defective speech. Early operations, and plans of operating which will not interfere with muscular action are, therefore, always to be advocated.

PAPER:

THE COSMETIC AND PLASTIC SURGERY OF THE EAR.

M. A. GOLDSTEIN, M. D.

Cosmetic surgery, employed in the correction of deformities or abnormalities of the external organs of the head, is a comparatively new field of work to which the oto-laryngologist has, as yet, contributed but little of his time, talent or technical skill.

This department of surgery has been for the greater part in the hands of the "advertising specialist" and "beauty doctor." The work of correcting featureal defects or deformities has received but little legitimate recognition in the nomenclature of classic surgery, nor has the necessity for such surgery been deemed sufficiently justifiable to engage the serious attention of the busy surgeon. A markedly crooked nose, or an irregularly enlarged ear, may not seriously handicap the functions or utility of the general economy, but the patient may be just as sensitive and conscious of such a defect and anxious for its correction as he would be to have a genuvarum straightened by the orthopedist, or a strabismus corrected by the ophthalmologist. This form of surgery is dignified, delicate and justifiable, and may call into service all the skill and technical ability at the command of the surgeon.

There are many forms and variations of the surgical technique employed in this work; the operations may be performed without serious inconvenience to the patient, and in most instances without a general anesthetic; complications and serious sequelae should be of very rare occurrence if the usual aseptic and surgical precautions are observed; the ultimate results are gratifying both to the patient and to the operator.

The correction of defects of the nose and ear is distinctly within the province of the oto-laryngologist, and it is our opinion that we may confer an additional boon on humanity by wresting cosmetic surgery from the hands of the quacks, where it has seemed

to flourish until now, and giving it a legitimate place in our earnest considerations and active work.

I have classified cosmetic and plastic surgery of the ear in three subdivisions:

(*a*) *The correction of defects and deformities of the external ear,* including the many abnormalities and congenital stigmata to which the human auricle is heir.

(*b*) *A discussion of the plastic technique, following the radical mastoid operation,* with special reference to the operative details in

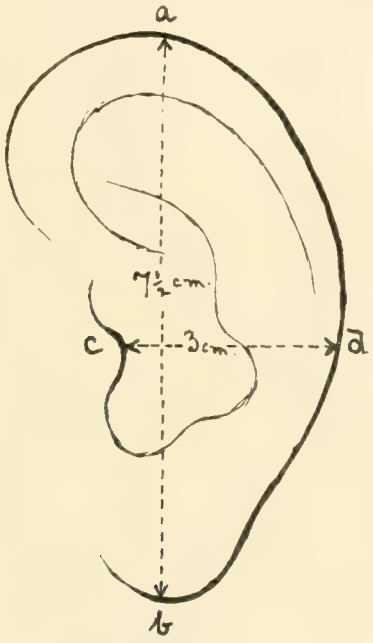


Figure 1.

Diagrammatic representation of normal ear measurements.

the formation of the various flaps, and a comparison of the special advantages of each of these plastics.

(*c*) *The correction and closure of persistent retro-auricular openings* following the radical operation, and a consideration of the various methods to perform such plastic.

There is perhaps no part of the human anatomy that is subject to greater variation in size, shape, and development than the external ear; to even the casual observer a considerable abnormality

in the size and shape of the auricle may make a definite impression of disfigurement. It may be impossible, therefore, to create any standard for a normal ear, or to empirically specify to what extent the deviation from such standard may take place without justifying surgical intervention. Until the technique of correcting deformities of the external ear is more accurately developed, and until this field of plastic surgery gains greater favor with the otologists, it may be well to confine our efforts and energies to the correction of those aural abnormalities where conspicuous deformity exists, and where the patient desires such correction because of the many personal embarrassments and comments to which such deformity may subject him.

An empirical standard may be established by measurements of the long and short axes of a large number of normal-shaped ears. The

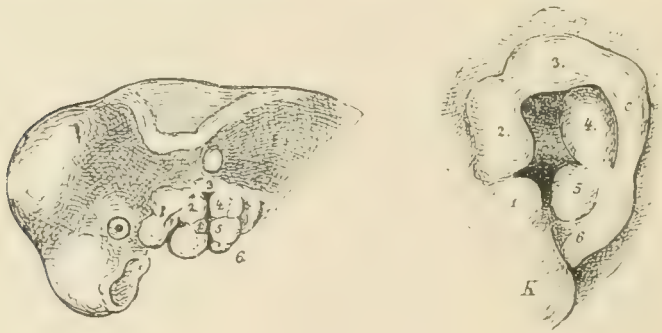


Figure 2.

Head of Human Embryo (His). Above the first branchial cleft lies the ear vesicle. Surrounding this cleft are the six tubercles of which the external ear is formed.

Human Embryo (His) showing the six tubercles of which the several parts of the external ear are formed. 1. Tragus. 2 and 3. Helix. 4. Anti-helix. 5. Anti-tragus. 6. Lobule. K. Inferior Maxilla.

long axis should be measured from the most dependent part of the lobule to the crest of the helix, as indicated in line *a b*, Figure 1. The short axis is taken from the inner curve of the tragus to the arc of the helix at right angles of line *a b* to line *c d*, Figure 1. These measurements are expressed in an average of a large series of ears as follows:

Long axis, or length of ear, 7 ½ cm. (3 inches); short axis, or width of ear, 3 cm. (1¼ inches.)

In the long axis all measurements exceeding three inches, or less than two inches, indicate an abnormal measurement. In the short

axis a width of less than one inch, or of more than one and a half inches, is regarded as abnormal.

Aural embryology offers the first evidence of the great variation in the construction of the external ear. Minot states that "before the end of the first month there appears around the external opening of the first gill cleft a series of six tubercles, two in front on the hind edge of the first visceral arch, one above the cleft and three behind it. A little later a vertical furrow appears down the middle of the hyoid arch in such a way as to mark off a little ridge, which joins on to tubercle 3, and descends behind tubercles 4 and 5. The second stage is reached by the growth of all the parts; diffusion of tubercles 2 and 3, and the growth of the ridge down behind tubercle 5, to become continuous with tubercle 6. After these changes it is not difficult to identify the parts.

"Tubercle 1 is a tragus; 2 and 3, together with the arching ridge, represent the helix; 4, the antihelix; 5, the antitragus; and 6, the lobule; the pit between the tubercles, the fossa angularis. During the latter part of the second month the ear changes in its proportion somewhat in the irregular development.

"The third stage begins at the third month; the upper and posterior part of the concha arises from the surface of the head and gradually but rapidly bends over forward, so as to completely cover the antihelix and the upper portion of the fossa-angularis. During this stage in mammals the assumption of the pointed form of the ear commences.

"The fourth stage commences at the fourth month, when the tubercles, which are now joined together by cartilages, commence to unfold and are complete by the fifth month. Finally the sixth tubercle develops to form the lobule."

Talbot, in his excellent monograph, "The Degenerate Ear," expresses the relation of embryologic development to aural deformities very tersely and clearly, as follows:

"This unfolding or development of the tubercles to produce the different portions of the ear and make it complete is not unlike the development of a flower from the bud. By this process may be understood how, if by malnutrition in one tubercle, or bud or another, or should there be a larger supply of nutriment in one than another, malformation of the ear would result. If an arrested development of all the tubercles should take place at any period from

the first to the fifth month of foetal life, the ear would resemble a semi-developed flower."

The anomalies of form to which the external ear is liable have been the subject of considerable anthropological research, and the impetus to this research was given by the keen observations and accurate conclusions of the eminent Italian criminologist and anthropologist, Lombroso.

About fifteen years ago Gradenigo created additional interest from an otological point of view, by the publication of his classic.

Figure 3.

Asymmetry of the Auricle.

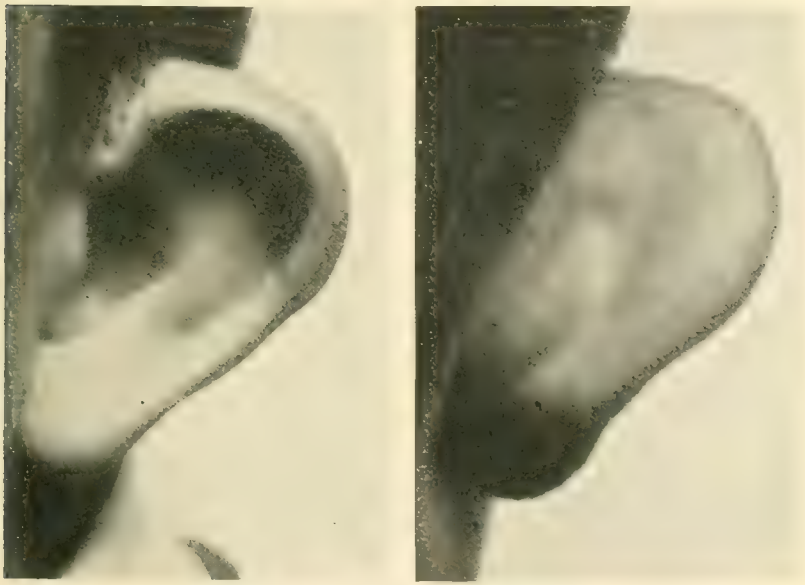
anthropological studies, "Ueber die Formanomalien der Ohrmuschel."

The most conspicuous of the anomalies as classified by Gradenigo are:

1. Macrotia and Asymmetry of the Auricle. Where the distinct variation in size and shape of the two auricles in the same patient exists, it has been found, with but few exceptions, that such asymmetry is associated with facial asymmetry. In this subdivision of asymmetry of the auricle will be found a large proportion

of the cases that come under our observation for legitimate correction.

2. Heterotopy. Cases of misplacement, displacement and abnormal insertion of the auricle are rare, and are to be found only in the most marked deviations of embryologic development and especially in certain monstrosities known as otocephali. A number of the earlier writers have reported such remarkable cases; Sebenicius reports an auricle inserted on the neck; Wolf, a case inserted on the shoulder; and Phelitz and Stark, an auricle located on the



Anterior View. Posterior View.

Figure 4.

Projecting Auricle.

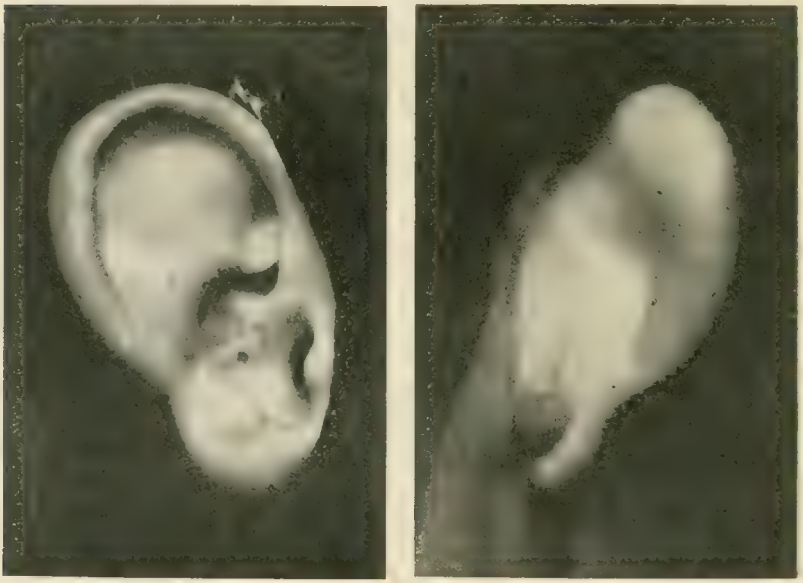
cheek. This class of cases is hardly to be considered within the field of plastic surgery, unless it were possible in these rare instances to transpose the auricle entirely to its proper position.

3. Adhesion of the posterior surface of the auricle, in whole or in part, to the head. These conditions, as anomalies, are extremely rare, and find no definite embryologic significance.

4. Projecting auricle. (Prominent ear.) The auriculo-temporal angle, as observed by Frigario, measured between the side of

the head and the posterior surface of the auricle, is an acute angle in the normal ear. When, however, the auricle stands at right angles, or in even more exaggerated forms at an obtuse angle, it assumes the characteristic appearance known as the projecting or prominent ear. This abnormality of position is perhaps the most frequently observed of all deformities of the external ear, and has both an important embryologic and anthropologic significance.

When the ear is thus unduly prominent, especially when observed in early youth, its surgical correction is simple, the result



Anterior View. Posterior View.

Figure 5.

Absence of Antihelix, Projecting Ear and Redundancy of Concha Cartilage.

satisfactory, and the patient saved much embarrassment. This deformity predominates in men.

5. The pointed ear. (The Darwin tubercle and the Satyr point.)

The Darwin tubercle results from an incomplete involution of the helix; the Satyr point occurs in quite a similar manner on the superior border of the helix.

When either of these deformity areas of the helix is marked they may be promptly corrected by a slight plastic on the posterior

surface of the helix, and the resection of a narrow wedge of skin; if necessary, even the projecting portion of cartilage may be pared off subdermally.

6. The Macacus ear. (Also known as Darwin ear.)

In this deformity there is a flattened or imperfectly defined helix, or there may be even an absence of a section of the helix.

7. Wildermuth ear. (Prominence of the antihelix.)

The antihelix may develop so prominently that it may overshadow the helix when the auricle is seen in profile, and the ear assumes a characteristic aspect. This anomaly has been observed more frequently in the female. In this malformation there is an exagger-

Figure 6.

Darwin Tubercle; Satyr Point.

ated absence of the normal shell-like concavity of the concha and pinna. In this form the anterior surface of the auricle appears flat, and in some cases the anterior surface is even convex.

8. Absence of the helix.

This condition is extremely rare, and would offer but little practical opportunity for plastic, for when there is a distinct absence of this delicately formed cartilage of the helix, antihelix or tragus, it

is a difficult surgical task to shape one, or to construct it from other or adjoining tissues.

9. Absence or exaggerated development of the lobule.

It is a well-known observation that deformities of the lobule are conspicuous and apparent even to the laity. Complete absence of the lobule is not infrequent, and produces a very characteristic appearance of the ear. In this class of cases the cartilage is distinctly bound round by integument, and the familiar pendent portion, comprising the lobule, is absent. Exaggerated development of the lo-

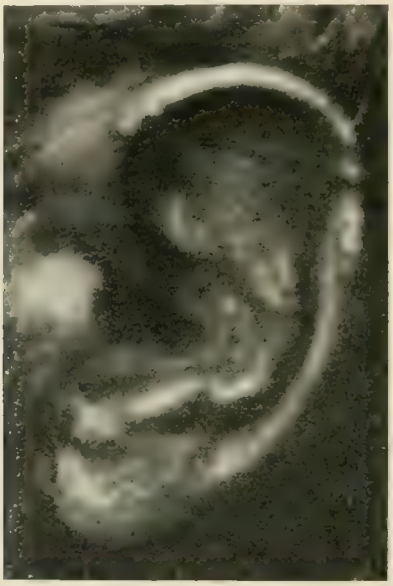


Figure 7.

Macacus Ear; Darwin Ear.

bule, either in length, in breadth or in thickness, is occasionally met with, and in such marked degree that a correction of this deformity offers a grateful field for plastic surgery.

10. Adhesion of the lobule. (Synechia.)

When the adhesion of the lobule is symmetrical, and the area of the external ear comparatively normal in appearance, it does not offer a conspicuous abnormality, but when there is a synechia of one lobule and the other lobule is normal, there is justification for its surgical correction.

This classification is a comprehensive one, and includes the major deformities to which the external ear is subject; many of the abnormalities, when sufficiently marked to warrant surgical interference, can be corrected by the several forms of plastic technique, which I will endeavor to describe:

PLASTIC TECHNIQUE.

The experiences which we have gleaned in the past few years in perfecting the details of submucous operative work on the nasal septum offer a new field in plastic ear surgery, for by applying the

Figure 8.

Wildermuth Ear: Prominence of the Antihelix; Absence of Lobule.

same principles which have been so successfully used in submucous work on the cartilages of the nose, to subdermal manipulation of the elastic cartilages of the ear, we are enabled to obtain satisfactory results in the correction of deformities, by measures which if formerly adopted would have been found impracticable and inadequate.

Correction of macrotia:—

Macrotia or undue length or width of the auricle may be such a conspicuous deformity that the patient will be glad to find relief

for this condition, providing the reduction in the size of the ear can be effected without much pain, loss of time, or perceptible scar. The operation which seems to have gained some favor for the cosmetic correction for this deformity, and which is occasionally briefly described in the modern text books on surgery, is illustrated in plate I (figures 1, 2 and 3).

An incision is made through the entire thickness of the auricle, from *a* to *b* (figure 1); the upper incised segment of the pinna is pulled downward over the lower segment, and with this traction

Figure 9.

Absence of Helix.

the operator gauges the amount of tissue necessary to be removed at the line *d b* (figure 2), to correct the deformity. An incision is made from *d* to *b*, and the overlapping triangle of tissue, *d b c*, composed of the cartilages and of the anterior and posterior surfaces of integument, is entirely removed.

The axis of the helix at *d b* is now seen to be shorter than the axis of the helix at *a b* of the lower segment. To obviate this, a narrow, wedge-shaped segment, *x y z*, is now removed, so that the line *d b* and the line *a b* may be of the same length, and the edges of the helix at the points *a* and *d* are in one continuous curve.

The edges *d b* and *a b* are now united by sutures alternately from the outer and inner surface of the ear, and a small suture may also be introduced, uniting the small wedge-shaped incision from *x* to *z* (figure 3). This technique will reduce both the length and the width of the auricle to such a size as the operator may elect.

The objections which may be offered to this method of operation are, that some scarring will result, both to the anterior and posterior surface of the auricle, and that the complete resection of the

Figure 10.

Abnormal Length of Auricle and of Lobule.

cartilage, without furnishing any underlying support for the newly constructed resected area, is a weak surgical principle. The cartilages of the ear are too poorly nourished, and the healing of such a wound will take place mainly in the thin integument, and will leave a permanent weak spot at the lines of incision through the cartilage.

Author's operation for correction of macrotia:—

To overcome these objections I have devised an operation which is simple, safe, and surgically sound. The principle of this technique is that of separating the cartilage from its perichondrium and skin covering, both anteriorly and posteriorly; of constructing flaps of cartilage to meet the requirements of the case in hand; of sliding the two cartilaginous flaps over each other; to effect a firm junction at the area of resection; and to do all of the operative manipulations on the posterior surface of the auricle, so that when the

Figure II.

Abnormal Width of Lobule.

single external incision is healed, an almost invisible linear scar on the posterior surface will be the only evidence of the operation.

The curvilinear incision *a b* (plate II, figure 1) is made on the posterior surface of the auricle, almost corresponding with the natural ridge of the antihelix anteriorly. The convexity of this curved incision line presents outward towards the rim of the helix. The flap is shaped so that when it is dissected upwards and over toward the mastoid area, the largest possible surface of cartilage is exposed. The dissection of the integument flap and perichondrium from the

cartilage is simple, and as the adhesion of the perichondrium is not firm, it can be quickly done.

When the cartilage is cleanly exposed, an incision in the line *c f* (figure 2), curving inwards, almost at a right angle at points *c* and *f*, is made through the cartilage, care being taken not to wound the underlying integument which forms the anterior covering of the auricle. With a narrow, blunt elevator, such as is used in the resection of the nasal septum, the two cartilaginous flaps at *c d* and *e f* are now carefully elevated from the underlying perichondrium to a sufficient breadth to admit of their manipulation, and to per-

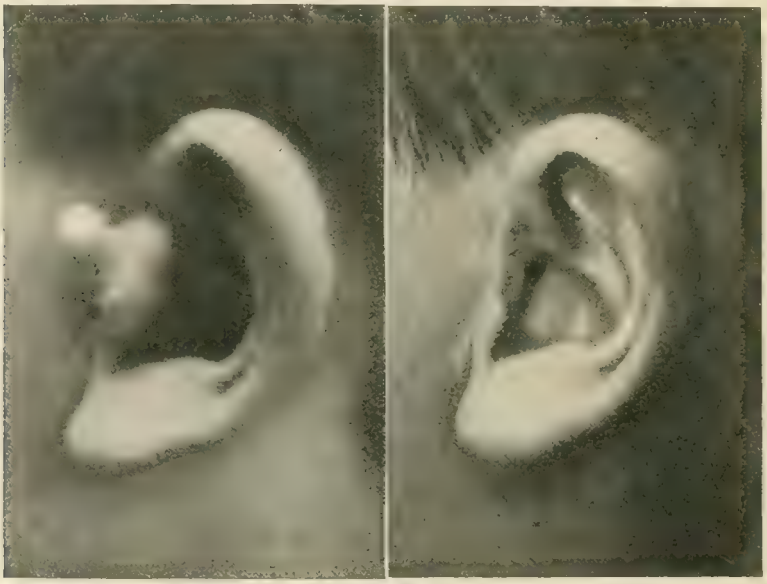
Figure 12.

Synechia or Adherence of Lobule.

mit the flap at *c d* to be superimposed upon the flap at *e f*, when brought together by a series of two mattress sutures, each suture being passed twice through each of the cartilages. When gentle traction is applied in tightening the sutures, the two cartilaginous flaps will be drawn over each other and the width of the auricle reduced in the same proportion as the sutures are introduced from the edges of the respective cartilaginous flaps. The sutures are applied by first passing a sharply curved needle, armed with thin chromatized catgut, through a point at the

upper end of flap *c d*, then downward through a point just opposite in flap *c f*; then from below upward through flap *c f*, then over and under through an opposite point in flap *c d*.

Another suture is similarly placed in the lower part of these flaps. The cartilage is tough, and will stand considerable traction when the sutures are brought together. The sutures are then tied over flap *c d* (figure 3), the redundancy at the edge of the integument flap *a b* is then carefully cut away and the integument stitched in position in the original line of incision (figure 4). With the trac-



Before Operation. After Operation.

Figure 13.

Supernumerary Tragus.

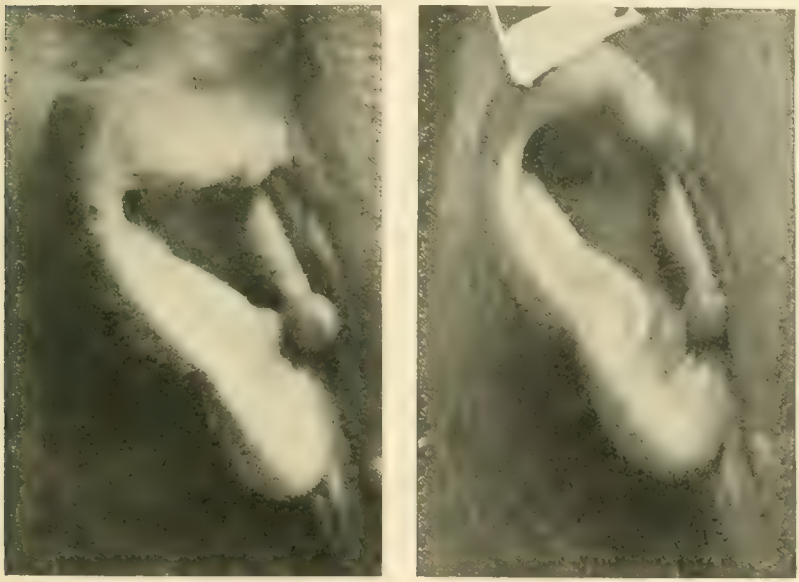
tion produced by the overlapping of the cartilaginous flaps, some slight wrinkling or puckering may at first appear on the anterior surface of the auricle, but if the anterior perichondrium and integument have been liberally elevated from the cartillage, this wrinkling of the integument will disappear with the healing of the wound.

I have simply described the technique for reducing the width of the auricle by modifying the line of incision and shaping the curve of same to conform to the area where reduction of length or width of

the auricle is desired. This operation may be utilized for the correction of any form or extent of enlarged auricle.

An examination of such an ear after the wound has healed will show the reduction in the length or width of the auricle, shaped according to the judgment of the operator, and will present a reinforced and overlapping cartilaginous surface at the area of resection and an almost invisible linear scar on the posterior surface of the auricle. The anterior surface of the ear shows no participation in this operation.

I believe that this method of operation, with such modifications as may suggest themselves to the operator, in the re-shaping of



Natural Position. Helix stretched upwards showing deep fistula and undeveloped tubercle.

Figure 14.

Multiple Deformity of Auricle.
(Case of Dr. J. B. Shapleigh.)

each given case, may be utilized for the cosmetic correction of all forms of macrotia and abnormally enlarged ears.

The correction of the prominent ear:—

The correction for the prominent or projecting ear has been, perhaps, the most popular and most frequently undertaken of any

in the field of cosmetic surgery of the ear. Where this deformity is very apparent, and where the auricle projects at an unusually prominent angle, the cause may be ascribed to an undue redundancy and elasticity of the cartilages of the pinna. In some of the chapters on plastic surgery in surgical text books the correction of this deformity has been dismissed with but a few words, and the operation suggested for this correction has consisted simply of an incision of an elliptical section of skin over the posterior surface of the auricle, close to the insertion line of the auricle to the head, and the

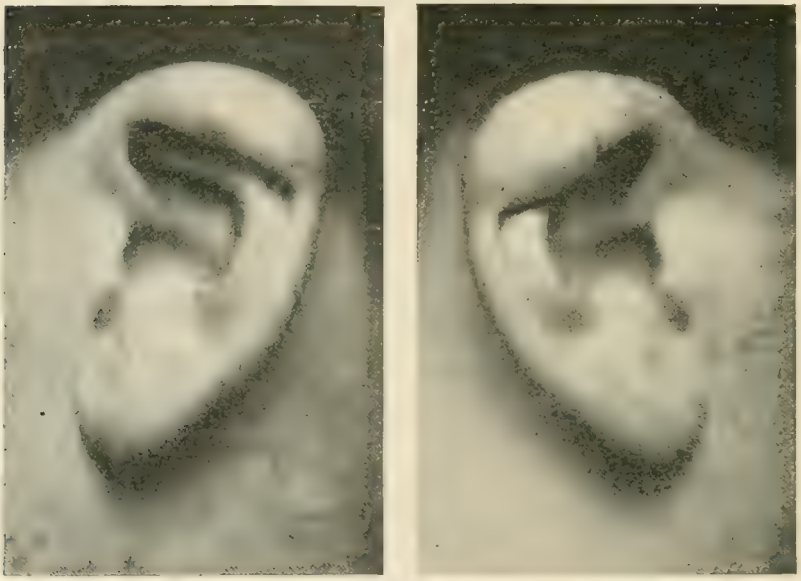


Figure 15.

Redundancy and Deformity of Helix.

operation completed by a line of sutures of the two presenting integument flaps. In the unduly prominent ear there is a preponderance of cartilaginous surface, and this simple form of operation is insufficient.

We again draw on our experience in the plastic surgery of the nasal septum where it has been definitely determined that the correction of deflected septa is not overcome until the redundancy of cartilage is removed. This same principle must apply in the correction of the prominent ear; the biconvex lens-shaped section of

skin, *a b c d* (plate III, figure 1) is removed; a narrow elliptical section of cartilage, *e f* (figure 2), is now carefully elevated and excised; the cartilaginous edges, *e f*, are drawn to the periosteum, *g, h,* by three or four thin catgut sutures (figure 3), and the two skin-edges *a b c* and *a d c* are sutured (figure 4) in position.

The principal object in this simple operation is to remove the redundancy of cartilage and reinforce the skin traction by anchoring the underlying resected cartilage to the periosteum over the mastoid area.

Figure 16.

Deformity of Auricle, produced by Perichondritis and Necrosis of Cartilage following the Radical Mastoid Operation.

Correction of the misshapen helix:—

This deformity rarely assumes sufficiently prominent proportions to warrant surgical intervention, even for cosmetic reasons, but at times a slight operation may be grateful to patients, especially women of comely appearance, where such deformity offers much embarrassment. In many of these cases I have found a redundancy and curling over of the skin covering the helix rather than a hyperplasia of the cartilage comprising the helix. In this class of cases it is simply necessary to make an elliptical resection of the skin

over the posterior area of the pinna, near the helix, thoroughly detach the integument from the cartilage of the helix, so that it may be easily drawn backward, and suture the two surfaces of skin with a small needle and thin black silk. An adhesive strip drawn from the anterior surface of the pinna over the helix and stretching backward over the posterior surface of the auricle, completes this plastic.

The correction of exaggerated size of the lobule:—

The preponderance of adipose tissue of the lobule either in length or in width produces a very unsightly ear, and when this is unilateral cosmetic correction is justifiable. As the lobule is constructed entirely of soft tissues and quite vascular, it may be easily

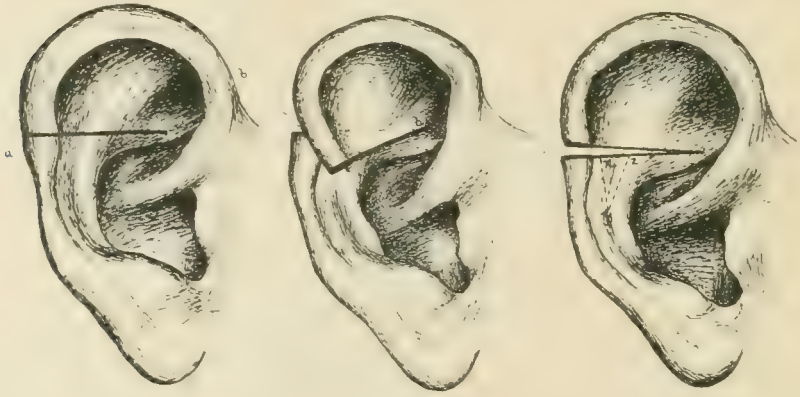


Figure 1. Figure 2. Figure 3.

Plate I.

Usual technique for Reducing Macrotia.

reduced in length or width by a simple resection of a wedge-shaped piece, and the approximation of the edge by one or two sutures; the parts are vascular and heal promptly.

The correction of synechia of the lobule:—

This abnormality in the shape of the ear is very frequently met with, and is at times sufficiently marked to cause the patient to ask for its correction. The following method is suggested to rectify adhesion of the lobule:

Raise the flap *a b* in front of the ear (plate IV, figure 1), with its base attached to the lobule, and dissect it up to a line corresponding with the normal perpendicular of the lobule. On the pos-

terior surface of the lobule raise the flap *c d* (figure 2), having its base attached to the extended insertion line of the auricle on the neck. Divide the lobule along the line *e f* (figure 3), check the bleeding by hot compresses or adrenalin; with the flap *a b* cover the exposed posterior surface of the lobule, and with the flap *c d* of similar length and shape the wound surface of the neck is covered. If the flaps have been carefully shaped and are nicely adjusted they may be held in position by aseptic adhesive strips without suture.

There have been other methods presented for operative plastic

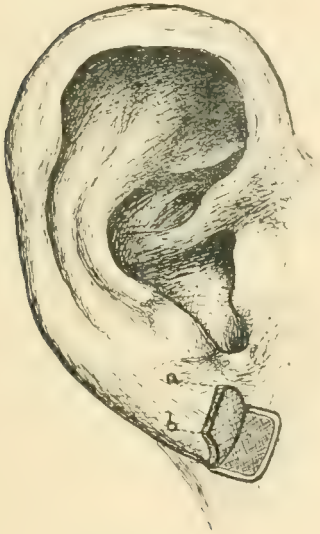


Figure 1.

Figure 2.

Figure 3.

Plate IV.

Radical Correction of Synechia of the Lobule.

on the ear, some of which appear impracticable; others too complicated to produce thorough coaptation of wound surfaces, and productive of unsightly scars; and some few even impossible. I have confined myself to a description and selection of such methods of operative technique as may be legitimately and safely carried out, and which will yield satisfactory results.

PLASTIC SURGERY OF THE AUDITORY CANAL.

Perhaps the most important use to which plastic surgery has been put in otology is the construction of the skin-flap shaped

from the membranous anterior canal wall, and utilized in lining the greater part of the excavated bone cavity, following the radical mastoid operation. The proper means of obtaining epidermization of this large bone cavity still forms one of the much discussed problems of the radical operation.

The history of plastic surgery of the mastoid dates back to 1899, when the Stacke modification of the Schwartze operation was first presented. The character of the technique of the Stacke modification demanded that the exposed bone cavity so produced be covered in some way, or reconstructed from some tissue to insure a permanent healing of the wound. In the Stacke operation, where the tympanic cavity proper is exposed and operated upon before the antrum is reached, where the membranous auditory canal is stripped from the bone, and where a considerable resection of bone is made, it is evident that some measure must be undertaken to cover the newly exposed bone surfaces.

It first occurred to Stacke to utilize the membranous auditory canal as an epidermal lining for the exenterated bone cavity, and the first plastic method associated with the mastoid operation consisted of simply splitting the plastic canal wall by a longitudinal incision in the median line, reaching from the fundus to the concha. This produced an upper and lower triangular flap, but because of the resiliency of the cartilaginous part of the canal wall and the insufficient size of the flaps so obtained, an improved plastic method was offered by Stacke, and later modified by Jansen, now known as the Stacke-Jansen plastic. (Stacke Plastic, plate V.)

The Stacke-Jansen plastic consists of a longitudinal incision through the superior wall of the auditory canal from the fundus to the concha. At right angles to this incision at the concha end a second incision is made, forming a rectangular flap, the full size of the posterior canal wall. This flap is pulled downward and backward to cover the lower area of the exenterated bone cavity. Jansen holds the flap in position by forceps or a narrow chisel, and introduces the gauze packing through the enlarged auditory canal, thereby holding the flap in its new position. (Plate VI.)

The Panse plastic is a substantial modification of the Stacke flap; the first incision is made longitudinally along the median line of the posterior canal wall, extending from the fundus to the concha; at the exterior end of this incision a perpendicular incision is made, forming an elongated letter T; or in other words, a longi-

tudinal median incision, with two right angle flaps, one ending in the superior line of the canal wall, and the other in the inferior line.

One flap is turned upward and lines the superior portion of the bone cavity; the second flap is turned downward and lines the posterior wall and floor of the bone cavity. This plastic admits of the largest possible area for inspection of the field of operation. Panse recommends that in the making of the flap incision a pair of forceps should be passed into and throughout the length of the external auditory canal, and the posterior wall divided while stretched over the open blades of the forceps.

Another procedure which has proven of much value in a large number of cases, where the resiliency of cartilage has interfered with the proper coaptation of the flaps of the bone surface, was suggested by Panse: when the T shaped incision has been made the right angle corner of each flap is lifted up betwen delicate forceps and the underlying cartilage and connective tissue is carefully cut away with curved scissors, producing thinner and much more easily adjusted flaps; these may either be sutured into position or be held in place by gauze tampons. (Plate VII.)

The Körner flap is the plastic of choice of the majority of operators to-day, because of its simplicity of technique, and because the single, tongue-shaped flap affords the largest area to cover the exenterated cavity. The Körner flap consists of two parallel incisions through the membrano-cartilaginous canal, one along the upper border and one along the lower border of the posterior canal wall. To facilitate proper coaptation and adjustment of this flap, especially where there is a redundancy of cartilaginous and subcutaneous tissue, the under surface may be very materially pared down, and the stiff cartilaginous sub-structure dissected away. Care must be taken in the preparation of this flap to make a thoroughly clean dissection so that no particles of poorly nourished cartilage remain for subsequent sloughing. (Plate VIII.)

An active enchondritis following the various plastic methods in the radical mastoid operation may take place and may give rise to most marked and irreparable deformities of the auricle. (Figure 16.)

Siebenmann has presented a modified plastic by which the median incision in the canal wall is extended upwards and downwards to form a triangular or Y-shaped secondary flap in the depth of the concha. This flap suffers additional difficulties in technique, and,

as a rule, the membranous canal is not large enough to construct a triangular flap of sufficient size and tissue to be of any practical significance. (Plate IX.)

The Ballance flap is a more recent modification, and is especially adapted to the radical mastoid technique as suggested by Ballance, together with his plan for subsequent skin-grafting. The Ballance incision is of the shape of a shepherd's crook. A median incision is made through the posterior canal wall, extended from the tympanic end to the concha, and ending in a semicircular incision encroaching far into the concha, the curve of the incision being downwards. This produces a large, irregular-shaped flap superiorly, and a smaller inferior flap. Ballance prefers to suture his flaps into position. Ballenger recommends an anchor suture, held in place by little rolls of gauze, as a modified technique, to hold these sutures in position. (Plate X.)

It may be observed that the majority of operators to-day prefer not to suture the flaps in the radical mastoid operation, but depend for the adjustment of same on dressings and gauze packs. By omitting the suture there is one less possibility of infection, and the time of the operation is somewhat shortened.

There are other modifications in the formation of the plastic flaps used in the radical mastoid operation, as suggested by Kretschmann, Neumann, Passow and others, but the principal modifications have been here enumerated.

PLASTIC SURGERY OF RETRO-AURICULAR FISTULA.

In a large number of cases where the radical mastoid operation has been performed, conditions may be present where it is unadvisable to close the entire posterior wound, and conduct the entire after-treatment through the enlarged anterior opening. Where cholesteatoma has been found, where extensive sloughing of the soft parts over the mastoid area has taken place, where bone necrosis has been active, especially when associated with syphilis tuberculosis or other constitutional dyscrasia, where dura or sinus has been exposed or opened, or where for any definite reason the operator finds it necessary to critically inspect the exenterated area during the healing process, the posterior wound must be carefully packed and allowed to granulate from the bottom, and the after-treatment conducted similar to the plan employed in the classic Schwartze operation. In this class of cases it will frequently be

found, where granulation and cicatrization have taken place, that a well-defined fistula, varying in shape, depth and diameter, has been established.

The disposal of such post-auricular fistulae is often a troublesome matter to the surgeon. It is a safe plan not to undertake the plastic closure of such fistulae until the operative field has been thoroughly healed and the parts thoroughly epidermized or cicatrized and freed from wound secretions.

The first description of plastic closure of retro auricular fistulae is presented by von Mosetig Moorhof (*Monatsschr. f. Ohrenheilkunde*, 1899, Heft 1). The operation is performed under careful asepsis and general anesthesia, a tongue shaped flap is made below the retro-auricular opening, the size of the flap constructed being somewhat larger than the fistulous opening. The flap is carefully dissected up, so that a smooth underlying wound surface may be exposed. An incision is next made round the circumference of the fistula, and a free and freshened edge is produced by knife and elevator. The tongue shaped flap is now lifted from its bed and inverted into the fistulous opening, care being taken to preserve the hinge-like flap in its integument attachment to the lower edge of the fistula. By the inversion of the flap into the retro-auricular opening the epidermis presents inward and the wound surface outward. The flap edges are now adjusted to the freshened and elevated edges of the retro auricular opening by four catgut sutures. The edges of the lower wound, from which the flap has been taken, are now approximated by two or three sutures. The raw surface of the flap presenting externally, is either covered with skin grafts or allowed to cicatrize, the author preferring the latter method. See plate XI.

Another form of plastic was suggested by Trautmann (*Archiv f. Ohrenheilk.*, Bd. XLVIII, Heft 1). Trautmann performs this operation by means of infiltration anesthesia. He creates two flaps of equal size, taking these from the lateral periphery of the retro-auricular opening; these flaps are crescent-shaped and measure four mm. at their widest point. The anterior flap includes the perichondrium; the posterior flap includes the periosteum; these flaps are turned inward to cover the post-auricular opening, and their edges approximated by four thin catgut sutures, as indicated in figure 2, plate XII. In placing the sutures the author emphasizes the practical value of passing the needle in and out of the same side, as he

finds it a much simpler technique, and can adjust the position of his suture points much more easily than by using the needle from one side to the other. The next step in the operation is to elevate the skin about the freshly made circumference, as indicated in figure 3, plate XII. Three sutures are now placed in position from side to side, and the skin drawn over the surfaces of the newly constructed flaps, and the edges approximated in the median line. The advantages claimed for this form of plastic are:

Before Operation. After Operation.

Figure 17.

Polyotia and Macrotia with Congenital Aural Fistula.
(Case of Dr. W. D. Black.)

(a) That it admits of the plastic closure of larger openings.

(b) That no wound surface remains exposed.

(c) That the reinforcement created by the underlying flaps and the overlying integument produces a firmer closure of the retro-auricular opening.

There are several points in criticism of these plastic operations which I desire to offer, and for which I present another form of operation, the results of which practically substantiate my position.

The retro-auricular fistula is the result of necessity, and not of choice. It occurs most frequently after extensive bone exenteration; it is produced by our reluctance to entirely close the posterior wound in order to keep the field of operation thoroughly under observation and control. Under these circumstances the wound heals more slowly, and frequently with extensive cicatrization, binding the dermal tissues tight to the underlying and frequently deeply excavated bone.

The clinical picture of such a retro-auricular opening is not often as simple and favorable to post operation plastic, as is presented in the technique of Mosetig-Moorhoff or Trautmann. Sometimes the cicatrized tissue is so extensive and the excavated bone area of the mastoid so deep that it is a practical impossibility to construct the flap of healthy skin below the retro-auricular opening, because no healthy skin can be found there. Often the stellate cicatrization spreads about the circumference of the retro-auricular opening so freely that it is not surgically possible to construct the crescent-shaped bi lateral flaps of Trautmann at a distance of four mm., or even of six or eight mm., from the periphery of the opening.

Author's Retro-Auricular Plastic.

The technique which I have carried out in a number of cases where such unfavorable conditions for plastic exist, is simple. I select the area where healthy skin joins cicatricial tissue, make an incision through the integument anteriorly to the perichondrium and posteriorly through the periosteum. The shape and length of this incision varies with the character of the cicatrix and the size of the wound. With a slender elevator, such as is used in the submucous resection of the nasal septum, the skin and perichondrium over the auricle anteriorly, and the skin and periosteum over the mastoid area posteriorly is elevated until sufficient tissue has been loosened to allow approximation of the two edges, with the least amount of tension.

The next step in the technique is identical with that used in the primary closure of the posterior wound following the radical mastoid operation. The two edges of the skin are carefully drawn together and held in position by the Michel metal clamps, the number of clamps used depending on the length of the wound. The underlying cicatricial tissue is not disturbed. The vertical incisions

made to relieve tension are allowed to granulate in their gaping areas.

Two vertical incisions, each of about fifteen to eighteen mm. in length, are then made at a distance of about ten to twelve mm. from the freshened margins of the wound, the anterior incision extending through the perichondrium and the posterior incision through the periosteum. The elevated tissue should extend to the lines of incision. By means of these incisions all possible tension when the wound edges are drawn together, is corrected.

This is a simplified technique, offering none of the disadvantages which are occasionally met in the methods of Mosetig-Moorhoff or Trautmann. In two or three cases the auricle has been drawn somewhat closed to the side of the head, because of the larger lateral diameter of the post-auricular fistulo.

The successful closure during the past five years of some fifteen or sixteen retro-auricular openings of various sizes justifies me in presenting this simple plastic technique.

The work that is represented in this paper is by no means exhaustive of the subject matter of cosmetic and plastic surgery as applied to the ear, but offers in a comprehensive way a critical summary of the most practical suggestions which have been gleaned from personal experience and from a careful search of modern surgical literature.

I believe this field to be a rational and logical department of surgery for the progressive, ambitious and skillful ear surgeon, if the cases are selected with care. If good judgment is exercised in the correction of marked aural deformities, the work of cosmetic surgery of the ear may be carried on with dignity, and may afford many opportunities for originality and a demonstration of excellent technical ability.

The plastic surgery of the ear as applied to the mastoid operation and to the conduct of retro-auricular wounds is a classic field, and our future attainments in this important chapter of aural surgery will depend largely on our perfection of the plastic work associated with the mastoid operation.

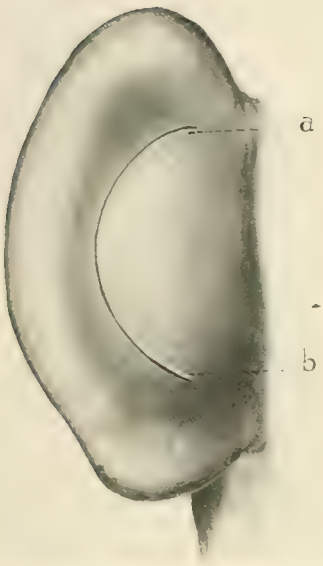


Figure 1

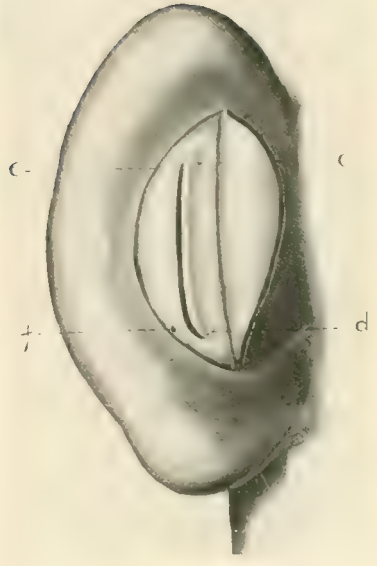


Figure 2.

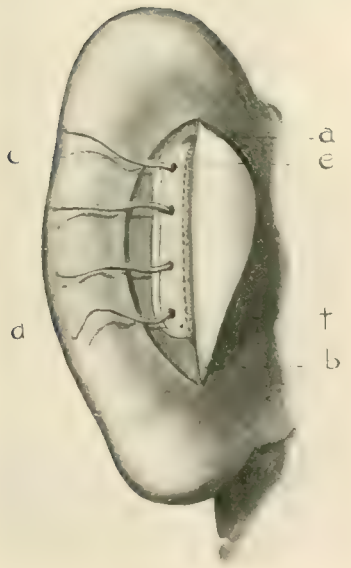


Figure 3

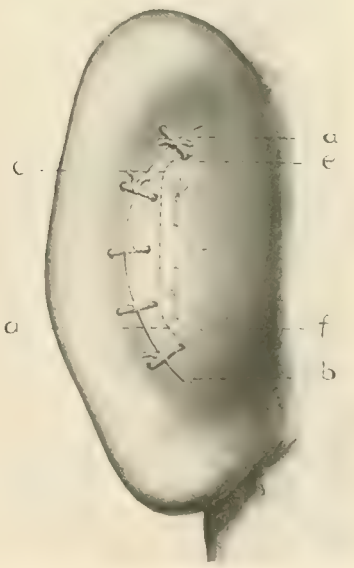


Figure 4.

Plate II.

Author's Operation for Reducing Macrotia.

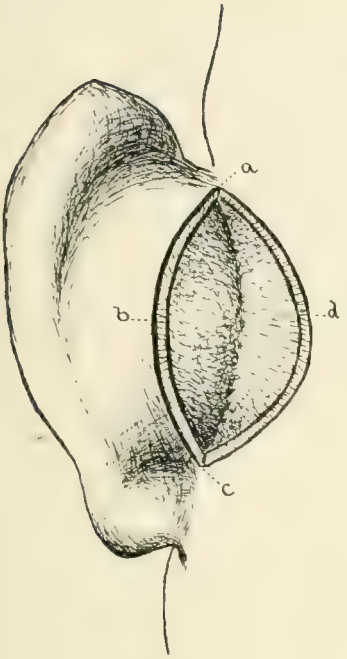


Figure 1

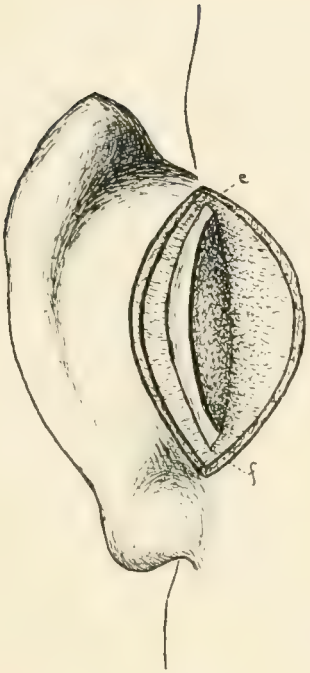


Figure 2.

Figure 3.

Figure 4

Plate III.

Author's Operation for Correction of the Projecting Ear.

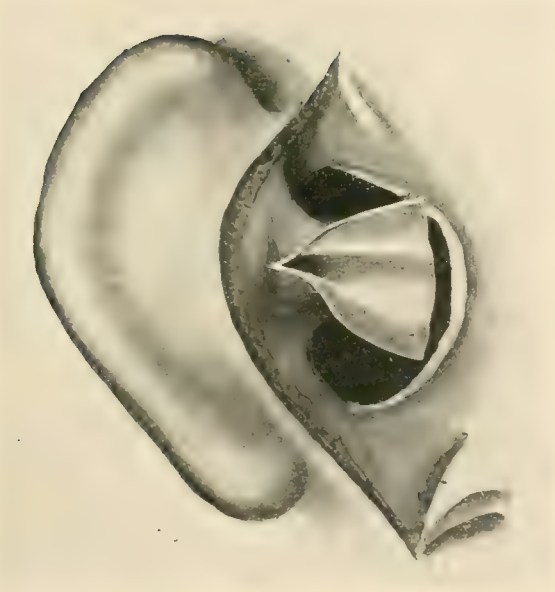


Plate V.

The Stacke Plastic.

Plate VI.

The Stacke-Jansen Plastic.

Plate VII.

The Panse Plastic.

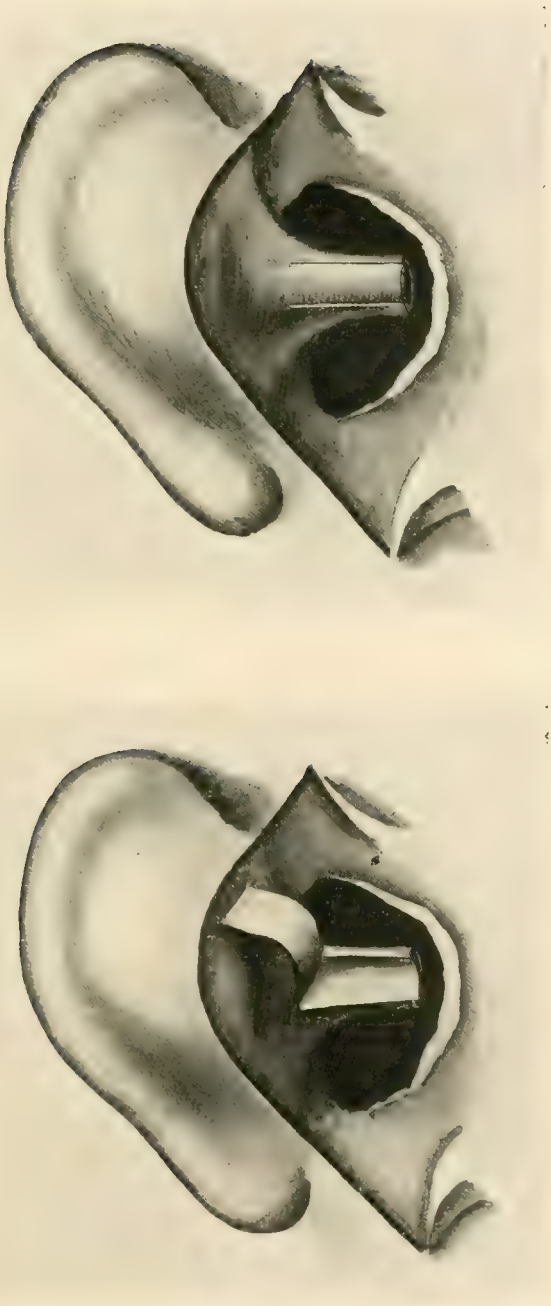


Plate VIII

The Korner Flap.

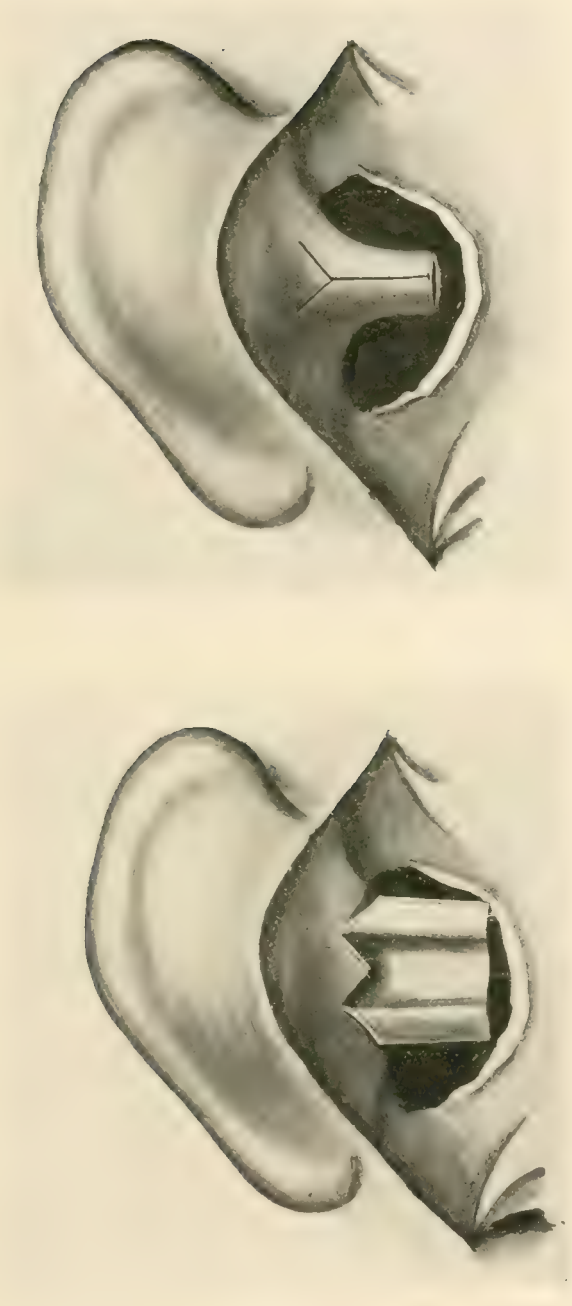


Plate IX.

The Siebenmann Flap.

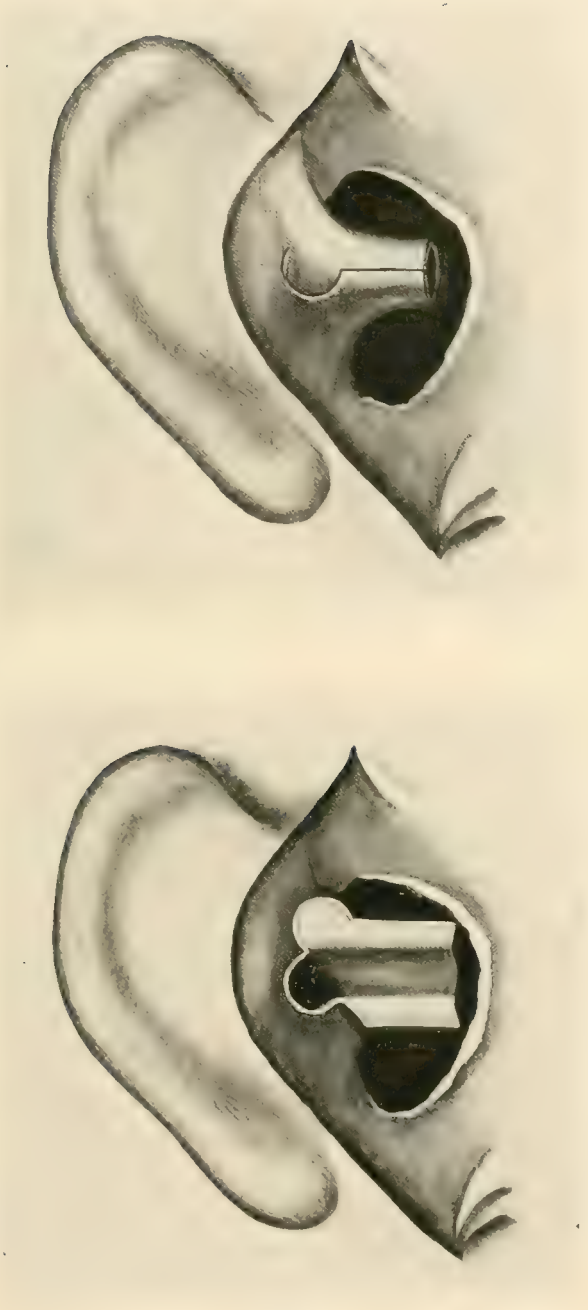


Plate X.

The Ballance Flap

Figure 1.

Figure 2.

Figure 3.

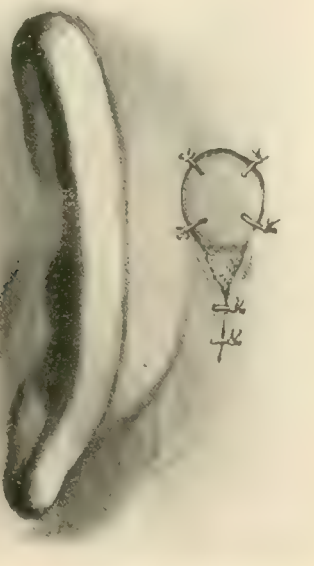


Figure 4.

Plate XI.
The von Mosetig-Moorhoff Operation.

Figure 1.

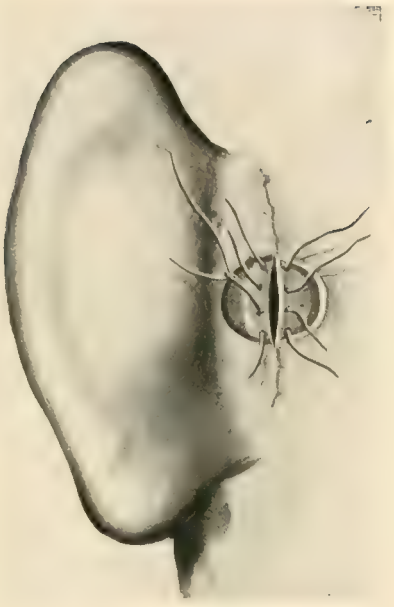


Figure 2

Figure 3.

Figure 4.

Plate XII,
The Trautmann Operation.

Figure 1.

Figure 2.

Plate XIII

Author's Retro-Auricular Plastic.

DISCUSSION OF SYMPOSIUM ON COSMETIC AND PLASTIC SURGERY.

DR. WILLIAM L. BALLENGER, of Chicago, confined his remarks to two points of technic which he had employed with satisfaction. First, referring to the "hook nose," mentioned by Dr. Beck, he had recently had a patient with combined humped and hooked nose. The hump portion he removed by the subcutaneous method, perhaps the exact method employed by Dr. Roe, though he had not seen the latter's operation. After freely elevating periosteum and skin he introduced the reverse chisel and shaved off the lump. He then corrected the hook by dissecting out a V-shaped portion of the cartilaginous part of the septum, taking out the entire thickness of the septum, leaving only the skin. Having done this he applied adhesive strips to the nose. The nose, instead of hanging downward, had the proper amount of extension, and the patient was very well satisfied with the result. The operation was done entirely by the subcutaneous or intranasal method which was not difficult of performance. Referring to the injection of paraffin, he had had one case in which the paraffin became dislodged after operation. He did not know how soon after the operation the displacement occurred, as he did not see the patient for a year following the operation. He did not remember the melting point of the paraffin. He injected the paraffin from below upward, which is contrary to the advice given by Dr. Smith. He had always done this, which, perhaps, accounted for his dissatisfaction with this method. He was indebted to Dr. Smith for his explanation of this point in technic.

DR. S. J. KOPETZKY, of New York City, reported the case and presented photographs, of a prize fighter who, after a blow on the ear during a bout, had an infection of the cartilage of the external ear. The case came under observation at the Manhattan Eye and Ear Hospital, New York City, and an incision was made in front, evacuating the pus collected underneath the skin. For about three months the cartilage continued to suppurate in spite of all attempts to stop it, until finally a great part of it had sloughed away, leaving a loose bag of skin which dropped forward, making a typical "dog ear." The subject was finally subjected to operation with a view to remedying the cosmetic effects. Numerous small elliptical pieces of skin were taken from the anterior side in order to attempt to reproduce the folds of the ear. From behind a tongue

shaped skin flap was cut and placed upon a denuded portion of the skull just behind the ear, suturing the tongue into place, thus holding the remains of the concha in position. Fig. 1 shows the concha before operation. The absence of cartilage is noticeable, the rim of the concha being folded in, and the bag of pus at the upper portion demonstrable at present. Fig. 2 is the end result after operation. During the operation some more of the cartilage of the concha was resected.

Dr. Arthur B. Duel called attention to the fact that unsightly deformities sometimes follow plastic surgery of the

ear by cutting across the crista helicis, instead of parallel with it, in the effort to get more skin. He had for a number of years been deeply interested in the subject of the plastic surgery of the ear and had believed that it should much more frequently be attempted by the aurist. In the majority of instances, the aurist hesitated to undertake these operations because of the danger of chondritis following interference with cartilage, and the further danger of this causing greater deformity than that which he was endeavoring to correct. By referring to the pictures presented it would be seen that the protruding ear was the result not so much of enlargement of the cartilage, but of a rolling out of the anti-helix.

It was possible in every instance to correct the most markedly protruding without interference with the cartilage, thus producing a better ear than would follow the removal of a wedge of cartilage. In no instance, in his experience, had the resilience of the cartilage been sufficient to pull the ear back.

Photographs were exhibited showing most markedly protruding ears which had been entirely corrected without any interference of the cartilage. The difficulty with the first cases had been that the amount of skin necessarily removed, to bring the ear back into correct position, had necessitated a cut so near the helix that the resulting post-auricular sulcus had been very shallow. He had therefore sought some method which would correct the deformity, without interference with the cartilage and still preserve the original depth of the post-auricular sulcus. He had accomplished this in the following manner:

The scalp was shaved as for a mastoid operation and the operative field rendered aseptic. The skin on the back of the ear was grasped with two pairs of mouse-tooth forceps in different positions and carried back to the scalp until the most advantageous points were determined. These were then marked by a tight squeeze of the forceps both on the ear and scalp, thus fixing the limits and direction of the subsequent incisions. Two incisions an inch through the skin and deep fascia of the scalp were then made, one along the post-auricular angle; another parallel to this and one half inch posterior to it. This band of skin was then dissected up thus forming a thick loop.

Parallel incisions through the skin, at a width corresponding to the marks made by the mouse-tooth forceps on the back of the ear, were then made at right angles from the anterior border of the loop up to the points marked near the helix (the points at which the pull should come to overcome resilience of the cartilage of the protruding auricle.) The band of skin between these incisions was dissected up, great care being exercised that the perichondrium was not injured. Two pairs of mouse-tooth forceps were then slipped under the loop and the end of the band of skin being grasped at each corner it was pulled back under the loop which acted as a fulcrum, until the ear was brought back into a correct position. With this held in place, the position on the scalp which the band then occupied behind the posterior limit of the loop, was outlined by the point of

a scalpel. In the same way, the area of skin on the band which lay under the loop was outlined. The band was then temporarily removed and the quadrilateral area of scalp posterior to the loop was dissected out. The area on the band of skin which was to lie under the loop was superficially denuded (this was best done by stretching it over a roll of gauze held in the left hand while the cutis was removed by a sharp scalpel in the right.—scarification was not sufficient.)

The band was then drawn under the loop again and held in place by suturing its end in the quadrilateral space made in the scalp behind the loop,—thus holding the ear in correct position. All lines of incision were then sutured together on the posterior aspect of the ear and on the scalp on the anterior and posterior borders of the loop. The lines at which the band passes under the loop were sutured superficially.

A few layers of gauze were laid between the ear and the scalp, a generous dressing applied and the ear held back by pressure. Stitches were removed in eight days. Pressure dressings should be applied for at least two weeks.

Photographs showing complete corrections by this method of marked deformities, still leaving the post-auricular sulcus of a normal depth were exhibited.

Dr. Frank Allport, of Chicago, said that the operation proposed by Dr. Duel was similar in its principle to his modification of the Panas operation for ptosis of the upper lid. Panas, of Paris, years ago proposed the making of a bridge flap in the eye-brow and a tongue flap in the lid and drawing the tongue flap underneath the bridge flap, and attaching it to the upper border of the highest incision. This, of course, shortened the lid. It had one defect, however, as it was impossible to produce a growing together of the skin surface of the tongue flap and the raw surface underneath the bridge flap. This always left a pocket or cul-de-sac which was not only disfiguring but which collected debris of various kinds and had to be cleaned out from time to time with alcohol, etc. Dr. Allport modified this operation some years ago by abrading, incising and scraping the skin surface of the tongue flap so that when the two were finally brought together a solid adhesion occurred which obviated the effect of the Panas operation.

With reference to the flap used after the radical mastoid operation, Dr. Allport preferred a modification of the Panas flap. In perform-

ing this portion of the operation he thrusts a sharp-pointed, double-edged knife from behind forward directly through the cartilage of the auricle, at a point corresponding as nearly as possible to the middle of the meatus. The knife appears a little back of the meato-conchal rim in order to enlarge the meatal orifice. This sharp knife is then withdrawn and another blunt-pointed knife made to take its place, as the see-saw movement necessary to finish the incision up and down would be likely to produce multiple punctures in the other side of the meatus, provided the use of the pointed lance-shaped knife was continued. The incision should correspond as closely as possible to the curve of the meatus and should extend up and down as far as possible. By making this portion of the flap operation at this time it is possible to control its size and shape better than if it were done after the longitudinal incision is made in the posterior tissue of the cartilaginous meatus. After this incision has been completed the spreader, or divulsor, is thrust into the cartilaginous meatus and the blades opened as wide as possible, in order to put the meatus on a tension. The lower blade of the divulsor can be used as a director and a blunt knife can now be introduced into the distal end until it reaches the incision previously made. Panas' original flap was made in the centre of the canal which made an upper and lower triangular flap practically of equal dimensions. Dr. Allport has found, however, that there never seems to be any trouble in healing over the floor of the bony cavity and therefore believes that it is best to give all the tissue possible to the upper flap in order that the roof may be speedily healed over. He makes a longitudinal incision as far down as possible so as to give all the tissue possible to the upper flap. The angle of the upper flap is sutured to the soft tissues underlying the skin with catgut and the cavity is then packed. He has found this modification of the Panas flap very satisfactory and finds that he is very seldom required to use skin grafts.

Professor Jansen, in discussing Dr. Goldstein's paper, said: "With the large persistent post-auricular fistulae, there is often combined a narrowing of the introitus of the external auditory canal. With Goldstein's method of closure of these fistulae the canal remains unchanged. Furthermore, the method does not seem to promise success in the cases of very large fistulae. For the past thirteen years I have been using a method, not hitherto published by me, which enables me to close the largest fistulae, at the same time

widening the canal. I make a circular incision, some distance from the edges of the fistula, about its upper, posterior, and lower margins, down to the bone. The skin is dissected toward the opening, for a distance of 3-4 to 1 cm., so that the edges of the skin may be reflected inward. By means of a vertical incision in front of the opening, the skin is dissected up from the posterior surface of the concha, also for a distance of 3-4 to 1 cm. A Korner flap is now made in the usual way, from the remainder of the posterior canal wall, and a portion of the concha. This flap is reflected backward, between the reflected edges of skin, which were dissected up from about the upper, posterior, and lower margins of the fistula, and sutured. In this way the fistula is closed in by a flap, which is epidermatized on its inner surface and raw on its outer surface. Over this area, the broadly mobilized posterior and anterior wound edges are drawn and sutured to each other and to the underlying tissues."

Dr. Wendell C. Phillips, of New York City, referring to plastic surgery for the correction of hare-lip and cleft palate, believed that Brophy's operation in infants offers the best hope of success. He had been watching Brophy's work and had seen a large number of his operations, and the point which impressed him most forcibly was the fact that in cleft palate there was no real loss of tissue. Another point which impressed him was that Brophy's early operation, being so nearly bloodless, obviates the danger which attends nearly all operations for cleft palate, viz., hemorrhage, and consequent shock.

Dr. Edward B. Dench, of New York City, called attention to the advantage of skin-grafting in plastic surgery of the ear. He had found it of distinct advantage to use grafts along the edge of the concha. The grafts had invariably taken, and the time of cicatrization was shortened. In cases of macrotia he had been able to make a fairly presentable ear by dissecting the skin away from the skull, grafting the entire denuded area, and then unfolding the auricle.

Dr. George L. Richards, of Fall River, Mass., described a method, which he had found very satisfactory, for operating upon crushed-in nose resulting from traumatism. Not uncommonly, such cases were treated by putting on adhesive plaster, which made the condition worse, and finally, when the crushed-in bone was firmly fixed, the patient would come to the rhinologist for operation.

Dr. Herbert E. Smyth, of Bridgeport, Conn., said that upon reading Dr. Brophy's description of his infant operation, he had received the impression that the approximation of the edges of the cleft was comparatively easy, especially after incising the malar processes.

Dr. Smyth unfortunately had had but one case suitable for this operation, an infant, three weeks old, with a remarkably wide cleft. He had had no difficulty in following the details of the operation as laid down by Dr. Brophy, but had found it impossible to approximate the edges of the hard palate, even after very free incisions in the malar processes.

Dr. Willis S. Anderson, of Detroit, Mich., was of the opinion that photographs of noses before and after operation did not always show the condition accurately, and in this connection called attention to the value of plaster casts in this class of work. The use of paraffin in the correction of depressions of the nose had the disadvantage that when the hollow was filled in by the paraffin, the nose was broader than normal. Paraffin should be employed only in cases which could not be corrected by plastic work inside the nose.

Dr. Robert C. Myles, of New York City, called attention to the value of the complete submucous operation involving the cartilage and the perpendicular plate of the ethmoid for the correction of flat, depressed noses resulting from severe traumatism. He had seen a number of cases where the nose had straightened up of itself after removal of the cartilage. By the use of a little paraffin, and by a certain amount of twisting and turning he had been able to get some remarkable results with plastic work.

Dr. W. W. Carter, of New York City, said Dr. Anderson had called attention to an important matter in regard to deformities of the nose. When the nose was broad and flat, it was absolutely unfitted to the injection of paraffin. The method which he had described for raising the bridge of the nose would seem better suited to this class of cases. Where the nose was very broad a portion of the nasal process of the superior maxilla could be chipped off with a chisel and used in building up the arch.

Dr. Beck, in closing the discussion, agreed with Dr. Anderson concerning the value of casts, many of which he had made. He took issue with Dr. Smith concerning the use of semi-solid paraffin, which would be absorbed to a great extent. He preferred Eckstein's method of injecting liquid paraffin, which would not be ab-

sorbed. He had employed paraffin injections in seventy-three cases. Dr. Ballenger's suggestion with reference to straightening the nose by the intra-nasal incision of the cartilage was entirely new. Senn had long ago excised the cartilage from the outside. The method described by Dr. Richards was not new, Roe and others having operated in this manner. Dr. Carter's method was excellent, and he would try it with the next suitable case.

Dr. Roe, in closing the discussion, said that for taking off superfluous bone by his subcutaneous method he had found the use of the saw far better than the chisel, as it is less liable to disturb the nasal bones and leaves a much smoother surface. This would especially hold true in the use of the curved chisel with the edge cutting forward, as proposed by Dr. Ballenger, for if the bones were not fixed firmly in position there would be more danger of displacing them with the chisel cutting forward than with the ordinary chisel cutting backward. In regard to shortening the nose by the V-shaped incision, Dr. Roe thought it far better to make the incision at the point of the greatest redundancy than high up near the center of the nose, as proposed by Dr. Ballenger. The success of all these subcutaneous operations for the correction of nasal deformities depended largely or almost entirely upon the judgment and experience of the operator as to where and how the incision should be made and the manner in which the work should be done. As cases vary so much in every particular, no prescribed rule could be followed, for the reason that the method which is suitable in one case is entirely out of the question in another case.

Regarding the methods described by Dr. Richards and Dr. Carter for correcting flattened or depressed noses by forcibly raising the center, Dr. Roe called attention to the fact that in all these cases where the deformity is the result of traumatism there is displacement of the nasal bones outward, leaving the nose much broader than before. Therefore, in correcting these deformities, it is necessary, in order to restore the nose to its normal width, to take off these bony ridges found on either side. In attempting to restore the symmetry of the nose by raising it in the center alone, it was necessary to raise the nose very much higher than it normally was in order to make it harmonize with its broader base. During his earlier experience in this work Dr. Roe had attempted this plan, and had devised special instruments for forcibly breaking and raising the center of the nose in these cases, but without success. It was

far better to cut off the projecting portions of these bones or displaced tissues and place them in the center of the nose than to elevate the nose to an undue height.

He had not observed the development of carcinoma as the result of the use of paraffin. His observations, however, regarding the use of paraffin were mainly confined to cases in which it had been used by others, for he had found it so much less satisfactory than his subcutaneous method for the correction of depressed deformities that he had not adopted it. He had also seen quite a number of cases in which the injection of paraffin had been most unsatisfactory, doubtless due in some cases, as Dr. Smith had said, to lack of skill on the part of the operator. In nearly all of these cases the paraffin had caused so much disfigurement, not only from unequal and undesirable distribution under the skin, but from marked and persistent redness of the skin, that the removal of the greater portion of the paraffin was necessary in order to relieve these conditions.

Dr. Goldstein, in closing the discussion, referred to a point to which Dr. Duel had taken exception. In the class of cases associated with macrotia there was distinctly a redundancy of cartilage which, from both the mechanical and the surgical point of view, must be disposed of. In the operation described by Dr. Duel, in which he had spoken of manipulating the auricle and drawing it back to the head, it was difficult to see how this redundancy of cartilage would be disposed of, unless he produced synechia of the two adjoining surfaces of cartilage. The removal of an elliptical section was much simpler. The modification of the Stacke-Jansen operation as described by Dr. Allport was practically the modification of Pascal, and had been performed for years. He had not included skin grafting in his paper because this was not necessarily a part of plastic surgery. The modification suggested by Dr. Jansen of Dr. Goldstein's operation for closure of post-auricular fissure would hardly be sufficient to make permanent closure without retraction. In two or three out of twenty cases operated upon by this method, he was able to place the knuckles of two fingers in the cavity, and yet he had no trouble in closing. Referring to the matter of plaster casts, he said it was difficult to get a cast of the ear on account of the softness and elasticity of the tissues. He described a method which he had employed satisfactorily by means of a composition used by dentists.

ORATION IN OTOLOGY.

"SURGICAL TREATMENT OF INFECTIVE LABYRINTHITIS AFTER FIFTEEN YEARS' EXPERIENCE."

DR. ALBERT JANSEN, BERLIN, GERMANY.

When the title of my paper was forwarded I was unacquainted with last year's transactions of the American Laryngological, Rhinological and Otological Society, and consequently the excellent papers upon this subject, especially that of Dr. Richards, were unknown to me. Had it been otherwise, I should certainly have selected another subject, being fully aware that, from my observations, not many new or original facts can now be presented.

May the fact of my having proposed the labyrinth operation, and of having carried it out for fifteen years, serve as an excuse in your eyes for the choice of this subject?

My observations are based upon upwards of one hundred labyrinth operations. In only two cases was the cochlea alone operated upon. In twenty-three cases the cochlea and vestibule were opened together and in the remaining cases only the vestibular apparatus. Only five of these cases followed acute middle ear suppuration.

Among those operated upon twenty-nine died, or twenty-five per cent, excluding four, in which death was not due to the labyrinth suppuration.

In sixteen cases the operation was performed after already existing purulent meningitis, and death could consequently not be prevented. In three others, death ensued undoubtedly from brain complications, likely cerebellar abscess, caused by the labyrinth infection.

I propose to arrange my subject synoptically into three or four groups.

The first group which, from its origin and course, is clearly distinctive from the remaining cases, comprises the infectious laby-

rinth affections caused by lesions of the labyrinth principally at the oval window.

In this first group of cases we are fortunately able to observe the origin of the disease. Therefore, the symptoms observed assume a decidedly diagnostic importance.

Injuries to the labyrinth are frequently caused by the radical operation. The horizontal semi-circular canal is the part most liable to injury; this injury is well known and comparatively frequent.

But of this kind of lesion I shall not speak here. I will only mention that it is generally recognizable at the moment of origin. It seldom leads to operative opening of the labyrinth or to fatal consequences.

In any case it is unnecessary to perform at once an opening of the entire vestibular portion on account of this lesion. Certainly the necessity may arise during the process of healing, but it is evidently exceedingly rare, and I can recollect only one case where the opening of the vestibular apparatus was afterwards necessitated.

We shall here treat of the lesions of the labyrinth, which are much more dangerous in their consequences; namely, the lesions of the fenestra ovalis and the luxation of the stapes.

This part of the middle ear is the one most exposed to lesion from curetting, and, excepting the horizontal canal, the part mostly endangered at the radical operation.

Sometimes the luxation, in the case of stapes disease, is very easily produced.

Through the accidental removal of the stapes the vestibule is exposed to serious danger, but the accident is more readily recognized and the danger thereby diminished.

As a rule, however, it is only a question of luxation of the stapes, of a partial rent of the ligamentum annulare, of an insignificant injury which, in opposition to the injured horizontal semi-circular canal, and to the removal of the stapes, we do not notice at the moment it occurs, and which we perceive afterwards only from the symptoms.

That we are dealing with a luxation of the stapes is at first merely a supposition.

Experience teaches us, however, that this supposition generally proves correct.

The great danger of the stapes luxation lies in the fact that under narcosis it occurs unnoticed and that infection of the vestibule immediately results.

In its consequences, as a rule, in the beginning, such an injury is far more disastrous than the opening of the semi-circular canal, while the initial clinical symptoms are usually far less alarming.

Half a day or more elapses before the first signs of labyrinth infection appear, and generally still more time is lost before they are properly appreciated.

When the accident occurs, let us say, from curetting in the after-treatment of radical operations, we can make the following observations:

Lesion of the labyrinth at once manifests itself in vertigo, disturbances of equilibrium and nystagmus.

It is true that these labyrinthine symptoms sometimes assert themselves only for a moment, coming like a flash of lightning and as quickly vanishing, often so suddenly that the nystagmus is no longer demonstrable.

Directly after the lesion, owing to the appearance of vertigo, the head jerks backwards or forwards, away from or towards the doctor. Sometimes both the head and the upper part of the body make this jerk. Not infrequently there is nausea or vomiting. For a few moments the patient is seized with violent vertigo; he is afraid of falling off the chair, everything seems to be turning round him or with him. On rising he seems unsteady on his feet, totters or falls.

Occasionally a subjective sensitiveness of the auditory nerve is present in the form of ringing or buzzing.

On examining quickly for nystagmus, it is usually found to be present, sometimes with quick, short, oscillating movements rather than with slow and long movements, and always strongest when the eyes are directed to the healthy side, or only in this direction. Vertigo appears sometimes only for a moment; sometimes it persists for several minutes, sometimes for a few hours.

Frequently, on account of the overpowering influence of pain, the patient is not conscious of the vertigo when it is of short duration, and does not complain of it.

The doctor mistakes these jerky movements caused by vertigo for a reaction from pain. Even after a few minutes the patient can often rise from his seat and move about again quite well. The

nystagmus often disappears too, and only returns as soon as the infection is established, but continues in case of labyrinthine hemorrhage. It is very easy to overlook the lesion with such feeble labyrinth symptoms. And now, when we have one out-patient to treat, we are confronted by a disastrous chain of circumstances.

The dressing of the wound is done in the usual way, and as the scraping out is often followed by hemorrhage, the packing is done tighter than usual for hemostatic purposes. This is another unfavorable moment, and facilitates the infection of the vestibule. The patient returns home; soon he feels worse and goes to bed. The following day he does not feel able to go out; he stays in bed; the dressing remains. The infection progresses and the case ends fatally.

A much more favorable view may be taken of the patient's fate if the damage is perceived at once, or if the patient is undergoing clinical treatment, and, consequently, an early ascertainment of the labyrinthine lesion is possible. The damage being at once perceived, a great deal may be done to render the complications less destructive in their course.

One must avoid any manipulation which is not absolutely necessary; powder with iodoform, push in loosely a piece of gauze for absorption of secretions, and attend to the constant discharge of secretion by frequent change of dressings.

With extreme care and absolute rest in bed, labyrinth infection is not infrequently prevented.

My cases of labyrinth injuries are nineteen in number:

1. Accidental extraction of the stapes	1
2. Luxation of the stapes (the diseased one) during dressing	1
3. Luxation of the stapes (the diseased one) during curetting	7
4. Luxation of the stapes (the diseased one) during the radical operation	6
5. Fissure through the horizontal canal	1
6. Lesions by galvano-cauterization*	3
	19
7. Infection by the curetting of a labyrinth previously exposed by operation	4

*These cases showed a slow course.

Spontaneous cure occurred in two cases; in the one, after scraping out, in the other one after accidental extraction of the stapes. In the latter case the symptoms of the labyrinth lasted for several days; they soon diminished and at last disappeared entirely with good hearing.

The second was affected with severe labyrinthine symptoms after scraping out, and subsequently for some weeks with severe symptoms of meningitis. At examination a good many years later the ear was deaf without caloric reaction. There was no disturbance of equilibrium.

Thirteen were operated upon with four fatalities, 37.7 per cent.

Life was saved:

1. In three cases with the complication of serous meningitis.

2. On account of the severe extension of the labyrinth disease in two cases.

It is interesting to know how long after the infection death occurred.

I. In the non-operated cases:

In two cases after seven days.
In one case after eleven days.
In one case after eighteen days (empyema sacc. endol.).
In one case after thirty-seven days (galvano-cauterization.)

II. In the cases operated upon:

In two cases after six days.
In one case after nine days.
In one case after eleven days.

III. In those cases which were curetted after opening the vestibule:

In one case after five days.
In one case after eleven days.

In half of all cases death occurred in from five to eight days.

Further, it is interesting to note on which day after infection the operation was performed.

I. In the cases resulting in recovery.

One after twenty-four hours.
One after twenty-seven hours.
One on the fourth day.
One on the sixth day.
One on the eighth day.

In two cases two months after operation and in the cases arising from galvano-cauterization:

One case the thirteenth day after infection.

One case the fifteenth day after infection.

II. In the cases resulting in death:

The second day after lesion, four days before death.

The second day after lesion, four days before death.

The third day after lesion, six days before death.

The fifth day after lesion, six days before death.

The tenth day after lesion, one day before death.

We see that in two cases the operation was performed too late, even on the second day after the lesion; also in another case, on the third day after the lesion, whilst still another case resulted in recovery, though the operation was performed four days after the lesion, and recovery has even taken place eight days after the lesion, as well as two months after. The longest period intervening between the operation and death was six days.

The question remaining for discussion is, whether in these cases of injury to the labyrinth death was accelerated or occasioned by the operation.

In the cases presented early for postmortem examination or for operation the tissues within the labyrinth always showed a hemorrhagic appearance.

The membranous labyrinth was deeply stained with blood, sometimes blackish red, always swollen, sometimes considerably so; the earlier seen the more hemorrhage it was. As a rule, the process was somewhat more advanced in the vestibule, sometimes when older it showed a greenish discoloration, occasionally also of the bone. The contents of the membranous canals were mostly light red, occasionally dark red.

In one case, indeed, there was after twenty-seven hours, a chocolate-colored mass in the horizontal semicircular canal, the vestibule showing a fresher red. In the early cases no pus whatever was found at operation or postmortem. In one case of postmortem examination after eighteen days the vestibule and cochlea were found filled with pus, also the much dilated saccus endolymphaticus. In cases that came under operation or postmortem later, great destruction was evident in the labyrinth.

These cases, if not operated upon, would probably have met the same fatal end. There is nearly the same period between death and

lesion in the operated and non-operated cases. Thus we cannot conclude that the death was accelerated or occasioned through the labyrinth operation.

Some of these lesions were rapidly followed by a very infective purulent meningitis. One patient showed the characteristic symptoms of meningitis two days after the injury to the labyrinth.

As only two out of the nineteen cases resulted in spontaneous cure, that is, 10.5 per cent, and even one of eighteen with luxation, that is, 5.5 per cent, we come to the conclusion that in the case of a determined labyrinthine infection it is better to open the infected part than to wait long for the labyrinth symptoms to increase or diminish.

But how are we to recognize labyrinthine infection?

By the rapid increase of the labyrinthine symptoms, often in connection with the signs of inflammation; headache, coated tongue, fever, reactive inflammation of the meninges.

According to pathological facts the question may arise whether there exists:

1. The purely traumatic injury only.
2. Simple irritation with or without injury, with or without hemorrhage (labyrinthinitis serosa hemorrhagica).
3. Infectious labyrinthitis.

In the first two conditions there is a good chance of spontaneous cure, while in the third there is a great tendency to go on to fatal ending. In the first group inflammation is wanting; in the second group inflammation is slight; in the third group inflammation and infection are severe.

In most cases we have an injury of the vestibular apparatus, and in consequence symptoms of vestibular irritation; that is, first of all, dizziness, disturbances of equilibrium, nystagmus. If these symptoms are produced, for instance by pressure upon the loosened or luxated stapes, the loosening of the dressing is sufficient to remove them. But if they continue, and even increase, they are proof of a severe and growing disturbance in the labyrinth that cannot be explained by the luxation of the stapes alone.

Sometimes the hemorrhage gives the explanation, but that is seldom the case. The hemorrhage may be caused by injury as well as by infection. If now pulse and temperature increase equally, the situation becomes clearer, but at the same time more dangerous. The higher temperature is hardly produced from the infected ves-

tibule or labyrinth, but in most cases (together with the pulse augmentation) by the reaction of the arachnoides. As soon as the temperature increases, be it perhaps only to 38° Cels. or so, for me the indication to open the labyrinth is absolutely given; but it would be wrong in case of no fever to neglect the other symptoms. We have seen that in one case the malady continued for eight or ten days without fever, the latter only setting in twelve hours before death.

Also in cases without fever it seems to me more desirable to perform the operation than to leave it as soon as the symptoms of labyrinthine infection are marked, persistent and on the increase, because it is much more probable that the malady will develop into an extensive labyrinthine affection, which would demand an operation later, perhaps under less favorable circumstances.

Even an exceptional spontaneous cure cannot turn me from this opinion.

How are the labyrinthine symptoms represented?

There are nystagmus, vertigo, disturbed equilibrium, as symptoms of vestibular affection; noises and the loss of hearing are the sign of the cochlear affection. In cases of lesions of the oval window all these symptoms of labyrinthine disease must be searched for generally in the early stage; they are not so evident as to obtrude themselves upon our notice. If the patient remains in bed after the operation these disturbances are not apparent. Nystagmus is slight but distinct. On looking straight in front the eye seems to rest quietly. On examining sharply with a bright light we observe, when the eyes are directed straight in front, a fine, slight, rapid rotary nystagmus, oscillating towards the healthy side. As soon as the eye passes the middle line towards the healthy side the twitchings increase, moving in long jerks and being either rotary or horizontal. The nearer the end-point the more extended the twitchings. They are often now so numerous that they cannot be counted, mostly remaining just about countable. In five seconds one may count five, ten or twelve oscillations, which are not regular and which sometimes cease.

In slight cases the nystagmus ceases if the eyes are directed toward the affected side. In severe cases it continues; and in other less severe cases it reappears, but then oscillating toward the affected side and more slowly. The growing intensity of the twitch-

ings is a symptom of the progress of the inflammation in the vestibular apparatus.

Lying in bed, dizziness is scarcely or not at all observed. The patient lies with closed eyes, his head pressed into the cushions, without moving. He avoids movements, which might produce dizziness, and so the patient, not being aware of the sense of dizziness, cannot complain of it. As soon as he is made to turn upon the injured side or to sit up, he will feel dizziness. Should the patient be requested to get up, vertigo would be more pronounced. When the patient is in bed disturbance of the equilibrium does not appear. If free sitting up in bed is impossible without effort and wavering, the disease must be a very severe one. In the early stage the patient is often able to walk without any unsteadiness. The patient himself may not have the slightest sense of disturbed equilibrium, not even when walking with closed eyes. If asked to place one foot before the other, there comes a slight wavering, sometimes only when the eyes are closed. With this experiment of standing with one foot before the other and the eyes closed, one is able to prove any degree of disturbance of equilibrium which otherwise is not observed.

The caloric reaction, which serves us so well in the diagnosis of labyrinthine diseases and gives us very exact knowledge of the degree of sensitiveness in the vestibularis, is really of no use here. In these cases where we must presume that there is an artificial passage, made by injury, leading to the labyrinth, we have to avoid syringing as long as we may hope to get a spontaneous cure. But putting in cotton with cold or hot water instead of syringing does not give reliable results. Moreover, we are observing the labyrinthine inflammation from its beginning, when there is not weakness of the vestibularis, and thus the symptoms are distinct and significant. The inflammation may rapidly progress toward the brain before the loss of the vestibular function. We may not forget to enumerate here headache and coated tongue as signs of an inflammatory process in the vestibule, although they may be produced by meningitis as well as fever. We do not doubt that in all these cases of labyrinthine injury in the early stage the vestibularis will very well react upon cold and heat.

In this early stage we are not often able to diagnose an infective meningitis. The symptoms are still deficient. By the lumbar puncture we may in the earliest stage find a minimal augmentation

of pressure, which, however, may quickly rise higher; a few times we found diplococci in clear fluid; once even after twenty-four hours; a little later, we find more leucocytes with it, and a turbid fluid. Among my cases, however, there are some of purulent meningitis without any considerable pressure augmentation, with no bacteria in the lumbar fluid, and even a clear fluid. That is why the negative result of the lumbar puncture can scarcely be used as a contra-indication against the operation. The operation ought to be done as soon as possible, in order to prevent the development of an infective purulent meningitis.

The following clinical picture is typical for a severe and progressive infection of the labyrinth:

The patient lies apathetically in bed, avoids moving his head or even raising himself in bed, refuses food and drink because of the inclination to vomit. He lies quietly with closed eyes, mostly on the healthy side; from time to time vomiting occurs, especially after taking nourishment or after moving. The tongue begins to get coated. Slight headache appears. On attempting an upright posture or on turning around he becomes dizzy. Sitting in bed is sometimes difficult or impossible. He gives the impression of being very ill. Nystagmus is manifest in the above-mentioned way, at first slight twitchings, increasing to quicker, more lengthened and irregular twitchings, rotary and horizontal oscillations, ten to twenty and more in five seconds, scarcely countable. They are very strong in the end position to the healthy side, continue looking to the affected side.

It is only when the patient gets up that disturbances of equilibrium are observed; at first by standing with feet placed one before the other, in severe cases with open eyes, or standing with feet together and eyes closed. At this stage I consider an operation indicated, although there may be no increase in temperature. In case of the slightest increase in temperature, if this is not due to the general wound conditions, the operation is imperative and must be performed immediately.

We have seen that in the opening of the vestibule the operation was performed too late even two days after the lesion. If the infection has penetrated from the vestibule to the arachnoids it is uncertain whether we shall be able to check it. Unfortunately, we have often a streptococcic infection. On the other hand, if the in-

fection has not yet penetrated the nerve canals, we may be able to arrest the process by a wide opening of the vestibule.

Symptoms of the cochlea are inconstant. Sometimes humming or ringing is experienced, frequently nothing.

The arachnoid membrane is exceedingly early affected by the inflammatory processes of the vestibule. In two cases lumbar puncture proved the existence of pneumococci in the fluid twenty-four hours and twenty-seven hours after infection. The pressure had risen only very slightly to 150 mm. In this early stage leucocytes are not yet present. In another case lumbar puncture showed a pressure of 220-300 mm.; it was transparent, free from bacteria and leucocytes. Once it was found transparent after forty-eight hours, and had only a pressure rising to 150 mm. In one case the liquid was clear even after four days, with a pressure rising to 290 mm. In a fifth patient, however, the cerebro-spinal fluid was turbid, but free from bacteria, with a pressure of 220 mm.

Just in the first few days when we are looking out for symptoms to fix the diagnosis and to decide the necessity for operation, the lumbar puncture seems to give us rather unreliable results, though in spite of this it ought to be done.

Fortunately, not all cases end so tragically, in which, directly after the operation, we notice those disquieting symptoms indicating labyrinthine affection. In my cases I have been able to include twelve with important labyrinthine syymptoms arising directly after the operation or for a few days later, dizziness, vomiting, nystagmus, disturbed equilibrium.

All these cases were cured promptly.

Moreover, in a number of cases—eleven or more—labyrinthine symptoms appeared during the first week after the operation.

With these patients the symptoms of labyrinthine irritation were of long duration, for weeks and even months.

Two possibilities are here presented. Either in all cases, or in a part of them, there is a luxation of the stapes and serous labyrinthitis or an infection of the vestibule induced by the injured cochlea, or there is a latent labyrinthitis, aggravated and made manifest by the operation or by the irritation accompanying the wound healing.

At the same time these cases prove that symptoms of labyrinthine disease do not give us a right immediately to operate in every case, or to oblige us to open the vestibular portion before satisfying

ourselves with the infectious and progressive character of the complication.

My numerous observations of the labyrinthine lesions have taught me that the greatest danger is imminent from the infectious disease of the vestibular portion and essentially from the vestibule itself. After having laid bare the vestibule we may generally consider the danger obviated. I usually combine the exposure of the vestibule with that of the ampullæ. But I do not pay any attention to the semi-circular canals, from which I fear no danger. There are no free pathways by which the infection from the semi-circular canals could get into the inner cranium. The smaller a canal is the sooner it is cured of inflammatory processes.

Although there exist some free channels leading from the cochlea and capable of conveying infection into the inner cranium, we have to deal with narrow canals similar to those of the semi-circular canals, and the danger becomes greater only when, by bony destruction, the narrow canals form a larger cavity with or without sequestra.

This seldom or never happens in cases of a rapid course. The infection arises in the vestibule, the disease extends into the vestibular apparatus, the symptoms are those of vestibular irritation, and if we interfere as soon as the welfare of the patient demands, a severe infectious process can scarcely have had time to develop in the cochlea.

These theoretical considerations agree with the experience we have had in such cases, and the results of which I have already stated.

The circumstances change when the infection is produced in the cochlea, or when the cochlear irritations have a share in the form of the disease known as humming or ringing in the ear, or noises of various kinds, or in processes having a slow course, where the development of the disease took many weeks, so that severe affectation of the cochlea may have taken place. Then the exposure of the cochlea may be indicated.

In the literature we find numerous proofs of this complication, as in the pages of Hinsberg, Freytag, Friederich, Heine, Zerony, Alexander, but I have noticed in these reports that rather too often the labyrinth infection is interpreted as a latent labyrinthitis, aggravated by the radical operation.

I come now to the second group.

Operations of the Labyrinth Resulting in Recovery.—There are sixty-two patients. The vestibule was opened sixty-three times, the cochlea eleven times; in one case the cochlea alone was opened.

Excepting the two cases the disease was always chronic middle ear suppuration which had caused labyrinth infection.

In the two acute cases a deep extradural abscess penetrated into the labyrinth at the posterior petrous plane.

Occasionally the affection was latent. The diagnosis was made either before operation, as was generally the case, or during the operation, through the evidence of fistulæ in the labyrinth wall or in the oval window, which was then usually found to be open.

Occasionally the labyrinthine disease evolves spontaneously during treatment, a long time after the mastoid operation.

In the cases which, before the operation, had shown severe labyrinth symptoms, these were, as a rule, greatly relieved after operation; often, on awakening from the anesthetic, the patients felt free from vertigo and much easier in the head, they could sit up in bed and move much better.

Inversely, those cases which presented slight labyrinthine symptoms frequently exhibited after the operation either slight or much stronger symptoms of nystagmus and vertigo for a short time.

In cases which, before operation, were free from vertigo and labyrinth symptoms, these latter occurred after the operation, now and then in a highly distressing degree.

Moreover, these appearances have been frequently met with by other authors. With striking frequency we read in the history of patients of the great increase in labyrinthine symptoms after the opening of the labyrinth. These appearances were manifest very strongly in one case immediately following the radical operation, when the fenestra ovalis was found open; there was a strong suspicion of traumatic affection recently caused or increased by probing.

The condition found at the operation one day later, however, contraindicated this, since the membranous labyrinth was glazed, swollen, not hemorrhagic.

In some few cases the labyrinth operation produced no changes whatever in the health of the patient; once I even found a child sitting up in bed and playing four or five hours after the operation.

In one case violent vomiting occurred at the moment of opening the horizontal semi-circular canal, and still another case experienced the sensation of being suspended in the air at the moment of striking off the lower vertical semi-circular canal, where the vestibule was opened. The operation was performed without an anesthetic.

In one case a discharge of cerebrospinal fluid came from the vestibule. On the other hand, we never observed any fluid discharge among this series of cases upon the opening of the cochlea; quite in contrast to the cases which ended fatally.

As complications we sometimes find sinus thrombosis without any connection with the labyrinth affection; once cerebellar abscess, twice purulent meningitis, in a number of cases serous meningitis.

Serous meningitis is a common complication in infective labyrinth disease. It is principally localized in the posterior cranial fossa, while the middle and anterior fossæ are free from it. In the puncture of the dura we found cerebrospinal fluid in great quantities only in the cerebellum.

Several times I could find proofs of an interesting affection upon the saccus endolymphaticus.

In several cases I found a circumscribed livid discoloration, about the size of a five-cent piece, on the cerebellar dura, from which the surrounding dura stood out rather sharply, owing to deficiency in livid discoloration and to its less moistened appearance. This area was close to the descending crus of the sigmoid sinus. I have often observed this phenomenon. There is no doubt, in my opinion, that it is a sign of an inflammatory process of the saccus endolymphaticus. The appearance depicted by this circumscribed livid discoloration in the vicinity of the sinus is a very characteristic one, and since recognizing this phenomenon and searching for it, I have come across it repeatedly.

It is a serous inflammation of the saccus which is in some cases productive of purulent inflammation.

When the radical operation has been performed, the closest inspection of the labyrinthine capsule is necessary.

In 1896 I reported upon 196 cases of defects of the semi-circular canal, which, with few exceptions, were located on the horizontal semi-circular canal. Since then I have seen 350 cases of infective

labyrinthine disease, of which again by far the majority were fistulæ of the horizontal semi-circular canal.

Although the fistula of the horizontal semi-circular canal is far the commonest, the fistula in the fenestra ovalis is by far the most dangerous. Exploration of the fenestra ovalis has only been systematically carried out by me of late years. Among the sixty-two cases resulting in recovery, the fenestra ovalis was found empty in twenty cases, and the horizontal semi-circular canal was mentioned as intact in ten cases; in several cases with transparent lumen.

The presence of fistula in the horizontal semi-circular canal is doubtless the chief pathway for the conveyance of infection to the labyrinth; but certainly not infrequently it must also be regarded as an escape from within.

When the invasion of the labyrinth occurs through the horizontal semicircular canal, there is present in many cases a circumscribed labyrinthine disease, the affection being limited to the semi-circular canal.

It is by no means always easy to arrive at a correct conclusion respecting the condition of the fenestra ovalis. The fenestra ovalis must be quite exposed for examination. The examination must be conducted with great precision and delicacy, lest we ourselves cause a lesion in or at the window.

We must also bear in mind that infection of the vestibule, if the vestibular wall is intact, may arise from the cochlea, which may be infected by an inconspicuous fistula in the promontory, or again, through a diseased fenestra rotunda.

It is now important to decide whether we have to deal with a circumscribed disease of the semi-circular canal, with a diffuse disease of the vestibular apparatus, or with a disease of the entire labyrinth.

Unquestionable cases have come under my notice in which the entire vestibular portion was attacked, but where the inflammation was restricted, for instance, to the horizontal semi-circular canal. The entire labyrinth can be attacked by a non-infectious inflammation.

The part attacked is, without doubt, either impaired in its functions or completely paralyzed. The cochlea would appear to become paralyzed in its functions more quickly than the vestibularis. The cochlearis is the more highly organized nerve, and the higher

the organization, the more delicate it is, and consequently, the greater the liability to decay and loss of function.

The following kinds of labyrinth affection may occur:

I. A serous, non-infectious inflammation of the entire labyrinth, or of separate parts of it, produced by the adjacent middle ear disease, with or without defect in the labyrinth capsule.

II. An infectious disease of separate parts of the labyrinth: (1) the cochlea; (2) the vestibule; (3) the semi-circular canal. There may be:

(a) A serous hemorrhagic infection.
(b) A purulent hemorrhagic infection.
(c) A purulent infection.
(d) Infection with necrosis.

The partially infective inflammation may be associated with a diffuse non-infective reaction of the entire labyrinth.

In every case of inflammation of the cochlea the function quickly ceases. If there is a serous affection or a quite slight infectious one, the process may result in recovery with a complete or, more probably, a partial restoration of function.

In a serous or slightly infective disease of the vestibular apparatus, the functional powers appear able to exist for a longer time. Here recovery may also be expected with complete restoration of the function.

If the patient's hearing is fairly good, we have, most probably, a circumscribed disease of the semi-circular canal.

More important and significant for the diagnosis are the phenomena of vestibular disease, signs of irritation or paralysis of the nerve. In the manifest cases the diagnosis may often be found at first sight.

Barany's method of caloric reaction most promptly acquaints us with the condition of the vestibularis and its functional power. We herewith test its sensitiveness by the application of cold and heat, by temperatures above or below blood heat. We use cold water of 12° to 15° Cels., and hot water (50°). With susceptible nerves slight differences suffice, with less susceptible nerves great differences in temperature must be applied. The results so obtained are especially valuable in those cases where the labyrinth symptoms have already disappeared, where the labyrinth disease is latent, or where it has resulted in spontaneous cure with paralysis of the nerve.

In syringing with cold and hot water, characteristic nystagmus and disturbed equilibrium combined with vertigo are produced. Nystagmus appears on the application of cold exactly as in the case of labyrinthine suppuration, on the opposite side, and, vice versa, on the application of heat, it appears on the same side, its intensity depending upon the temperature or the pathological condition of the nerve. We observe here all degrees of nystagmic movements from quite slight, scarcely perceptible, twitchings, to rapid, wide, wild and irregular rolling of the eyes. Similar to this are the disturbances of equilibrium, which sometimes do not exist at all, sometime only appear in slight degrees, and sometimes in so high a degree that the patient has no longer any control over himself. He is able neither to sit upright nor to stand, and in these severe cases the vertigo produced by rotary movement is extremely pronounced. Everything seems to be turning round him, and he himself collapses and falls. In slight degrees vertigo is sometimes completely absent.

The appearances produced by caloric reaction are perfectly in keeping with the phenomena of manifest labyrinthine disease.

Though it was known long ago that syringing with cold water produced vertigo, great merit is due to Barany for having followed up these symptoms most thoroughly, and for having constructed a method of practical use with the patient, which in latent cases of labyrinth disease enables us to arrive at a prompt conclusion of the condition of the vestibularis; but not of the kind of pathological alterations.

If no reaction occurs, paralysis of the vestibularis is proved. In most cases a purulent or infectious process in the vestibular apparatus will be the cause.

To answer the question concerning indications for operation the caloric reaction has to be used with precaution, and in those cases of very rapid development of labyrinthine inflammation, which are so very dangerous because of the possibility of a rapid infection of the arachnoid, it is also unsatisfactory. In these cases the caloric reaction has not yet disappeared at a time when an operation would give the best chance. In those cases where the operation allowed me to ascertain the condition of the inner labyrinth, caloric reaction was as follows: Four times negative, twice almost negative, neither vertigo nor nystagmus, but immediately after the test a severe fainting fit occurred, and the spontaneous nystagmus ceased

completely. Five times the reaction gave slight results, once with cold water, but no results from hot; four times a slight and three times a very marked reaction.

In those cases with marked reaction the operation gave the following result:

I. Very slight edematous swelling, semi-circular canal intact, fenestra ovalis empty and open. In a second case the semi-circular canal looked dark in its anterior portion. Fenestra ovalis empty, the membranous labyrinth in the cemi-circular canal and vestibule succulent and dark-colored. In the cochlea were granulations. In a third case (fistula in the promontory), much hemorrhagic fluid in the horizontal semi-circular canal. The contents of the vestibule dark red, hearing much lessened.

In cases with moderately strong reaction the result was as follows: First, the horizontal semi circular canal looked dark through the anterior crus, fenestra ovalis empty, in the vestibule a glazed swelling of grayish red color.

In cases where there is no reaction whatever the horizontal semi-circular canal appears dark in the forepart about the ampullar area. Fenestra ovalis penetrable by the probe. The membranous semi-circular canal is yellowish-red, in the area of the ampulla dark brown and represents here a stout cord.

Concerning the contents of the vestibule I could not come to a precise diagnosis.

The above phenomena further show that only the negative result gives a perfect proof; moreover, that we can find with a well-conserved reaction severe inflammatory alterations in the vestibule and semi-circular canal, which have often and quickly led to fatal meningitis.

The attack of caloric reaction indicates, moreover, a severe affection of the vestibule, but of what kind the changes are the experiment does not disclose. From the acute hemorrhagic swelling there would usually appear to be an important diminution, but sometimes only a slight one; with chronic glazed swelling and even pus in the vestibule, and, notwithstanding, caloric reaction can still occur in a more or less marked degree; there may be a cicatricial healing process or an ossification of the entire labyrinth or a complete suppurative decay; always the same result—loss of caloric reaction. For the diagnosis of an affection and the functionability of the vestibularis, the experiment is doubtless very valuable, but it discloses

nothing of a surgical advantage or of the character of the infection in question.

Do other labyrinth symptoms reveal anything similar, more important or less so?

The remaining symptoms in relation to the vestibularis consist of equilibrium disturbances, spontaneous nystagmus and vertigo, and are very significant, especially in combination with the caloric reaction.

We begin with nystagmus, a symptom which I have studied extensively in a great number and in a great variety of cases. But I will refrain from going into detail.

It is important for us to know that generally, but not always, severe nystagmus is a symptom of a severe affection of the labyrinth.

But what is the matter if we have but slight nystagmus or none at all?

In many cases where the vestibularis is completely or almost completely decayed, and consequently its function almost eliminated, we still observe spontaneous nystagmus. From my experience this kind of nystagmus is always directed to the healthy side, and is apparently produced by the cerebral center or the other vetibularis.

From the existence of only slight degrees of nystagmus we cannot conclude that there is only a slight affection of the labyrinth, and it would be quite a mistake to conclude that the vestibule is healthy if there is no nystagmus.

Just in those cases where labyrinthine infection is latent, where the nystagmus has subsided in consequence of disease of long duration in the vestibular apparatus, the result of caloric reaction is of great diagnostic importance and renders the affection manifest.

The disturbed equilibrium usually corresponds with the degree of nystagmus.

Since being in possession of the convenient and, on the whole, trustworthy method of caloric reaction, examination on the revolving chair, or with independent turning round, seems superfluous and is no longer used regularly.

The symptoms obtained from the disturbance of the vestibularis are, as already seen, numerous, and give a sharp clinical picture. The symptoms on the side of the cochlearis are far behind those of the vestibularis in their frequency or in their obstructiveness.

The symptoms of deafness become characteristic if they develop under our eyes, if we can witness the sudden change of Weber's experiment, the ascending of the lower tone limit, the descending of the upper tone limit, the loss of hearing for language.

The kind and degree of the equilibrium disturbances give us valuable signs for the intensity of the labyrinth affection, especially for its surgical importance, more than the lack of calorific reaction, the great value of which consists chiefly in making manifest a latent labyrinth affection. The more severe the labyrinthine symptoms are the more dangerous is the affection generally, and the more necessary is the opening of the labyrinth.

With the help of the improved diagnostic methods of examination and especially with the assistance of caloric reaction, we shall generally be able to diagnose the disease of the labyrinth before the operation. This is naturally of great value, for we can begin the operation with well planned intentions.

I come to the fatal cases:

In the cases I am now about to record death occurred within three to four weeks at the latest.

(1) The fatal complication was entirely independent of the labyrinth disease and operation in 4 cases.
Sinus thrombosis (with pyemia and abscess of the lungs) 1.
Meningitis tuberc. 1.
Arachnitis pur., induced by caries necrotica at the tip of the pyramid.

(2) Those produced by manipulating on the opened labyrinth:
Purulent arachnitis 3.

(3) Those produced by labyrinth operation itself:
Purulent arachnitis 2.
Empyema of the saccus endolymphaticus 1, not probably.

(4) Produced by the labyrinth disease:
Abscess of the cerebellum 3.
Empyema of the saccus endolymphaticus 2, accompanied in each case by pur. arachnitis.
Arachnitis pur. 18.

The important question now is in which cases death may be imputed to the operation.

One death by arachnitis was doubtless produced by the opening of the vestibule (the patient was a deaf-mute); there was ossification of the vestibular apparatus; in consequence of which the

inner auditory canal was opened instead of the vestibule, which resulted in a discharge of fluid. Death occurred nine and a half days later.

It is my opinion, the following death was also due to the operation though prior to the operation meningitis serosa was evident. On exposing the cochlea the inner auditory canal was opened, and after probing, a great discharge of fluid occurred. Death resulted in eight cases. The high temperature before the operation, owing to the meningitis serosa, diminished for a day and a half, and then increased on account of the purulent meningitis.

In both operations a technical fault was made.

The time between operation and death is nearly the same as in those cases where death was caused by injured vestibule.

A third patient now comes under our notice, who died eighteen days after the operation of suppurative meningitis, accompanied by empyema in the saccus endolymphaticus. At the radical operation a fistula in the vestibular wall above the fenestra ovalis had been overlooked. Immediately after the radical operation the labyrinth-symptoms, which had existed some days before the operation, increased, and two days later the labyrinth was opened, and so the fistula in the vestibular wall was discovered. The wound was in an excellent state. How can we explain the origin of these complications?

At first I refer to a case with injured vestibule. In the post-mortem eighteen days later, I found purulent arachnitis and em pyema of the saccus endolymphaticus. There is a remarkable agreement in both cases in the lapse of time between the lesion and the post mortem. In my opinion the infection of the saccus endolymphaticus occurred during the two days following the first operation with the increase of the labyrinth symptoms, and of course before the labyrinthine operation

The third and fourth cases, where death was caused by suppurative meningitis in conjunction with scraping out and Thiersch's skin transplantation, long after the exposure of the vestibule, prove that the operation itself does not dispel every danger, and that complications may be lurking in the subsequent treatment.

We have therefore five cases of death chargeable to operative manipulations upon the labyrinth.

It would be interesting to inquire which patients were free from meningitis during the time of operation. This question is diffi-

cult to answer. The lumbar puncture, of great importance for the diagnosis, has no absolute value. We have no absolute proof in those cases where we find a clear fluid, because in spite of this, there may already be a circumscribed and even rather extensive meningitis at the base. On account of want of time, I am sorry not to be able to answer this question more particularly.

It is certain that eight patients were suffering from meningitis already at the time of the operation, three patients apparently were not, but with two others there is a well founded suspicion of meningitis at the time of operation.

In the one case the wall of the auditory meatus internal was extensively necrosed.

In the second case there was an escape of fluid out of the cochlea.

In three cases the disease of the labyrinth appeared after acute influenza otitis, in the others it followed chronic otitis media.

Those cases resulting from acute otitis media were marked by severe labyrinthine symptoms. One of them, indeed, developed with lightning rapidity; within five days from the commencement of otitis media all was over. Severe labyrinthine symptoms were present in all three cases; vertigo, nausea, equilibrium disturbances and nystagmus; in one case of astonishingly long duration. In another case meningitis lasted a day and a half, in another from two and one-half to three and one-half days, with the third three and four days probably, and the labyrinth infection appeared two, four and one-half and nine days before death.

The infectious involvement with the acute otitis media has a more severe significance than with the chronic otitis media.

In the acute cases the membranous labyrinth was hemorrhagical.

Among the chronic cases this change was found only in one patient.

We now come to the second question:

Have the principles of the opening of the vestibule and the technical method of operating proved satisfactory, or are they to be modified?

I concluded from principle that in the majority of cases the vestibule portion is at first attacked, and is the most diseased part of the labyrinth, and the most dangerous, on account of its conduction to the meninges. On looking over all my cases I do not think that I should have obtained better results by opening the cochlea at the same time; it is certain, that in one case death was

caused by my opening the cochlea, although a technical error was manifest.

On exposing the labyrinth I generally use the method that leads to the desired result, by operating within the temporal bone without opening the posterior cranial fossa. Upon principle I only open the posterior cranial fossa, choosing the direction from behind forwards, if I have diagnosed an affection in the posterior cranial fossa, or supposed it to be present.

In one case, without doubt, I should have discovered and removed the caries and the sequestrum at the posterior petrous bony wall, and should possibly have found empyema in the saccus endolymphaticus. But whether I should be justified in every case in attempting the opening of the labyrinth from the posterior cranial fossa, seems to me doubtful, for the deep exposure of the cerebellum dura in the posterior cranial fossa is not absolutely without danger, and now and then I have experienced a rent in the dura, and thereby new channels for infection would be formed in the arachnoid.

ACUTE.

In the cases we have often spoken of an acute beginning, and mostly acute running labyrinth inflammation, but the middle ear suppuration was almost always chronic, seldom acute. In all my cases of labyrinthine affection I find only eleven induced by acute middle ear-suppuration. Four times death occurred, i. e., in thirty per cent of them.

Six of them have been operated upon the labyrinth, three too late because they were already suffering from meningitis.

With seven patients the labyrinth affection was started by a fistula in the semi-circular canal, in two cases without a fistula by opening the vestibule, with two other cases from the general clinical picture alone, which was very significant. On operating upon the membranous labyrinth it was always much hemorrhagically changed.

Three of them took the infection from the posterior temporal bony wall, after destruction of the vertical semi-circular canals by a deep extradural abscess. The frequency of this form in acute suppuration is astonishing. All these cases gave a picture of severe and dangerous illness.

From these cases we learn that severe labyrinth symptoms, with acute otitis media are a sign of a most dangerous affection, and that death may follow frequently and rapidly. With all of them

death came after two to six days from the beginning of the labyrinth symptoms.

In a fourth case during the healing of the wound most severe complications developed, an encephalo-meningitis serosa.

But in three cases we see that there is even a possibility of healing acute cases without operating upon the labyrinth.

As only a third of all cases was saved from death, and three of the cured patients had had intracranial complications, it is my opinion that we ought to open the labyrinth, if severe labyrinth symptoms of longer duration than twenty-four hours appear, following an acute suppuration of the mastoid process after the operation and with free passage for pus.

INDICATION AND PROGNOSIS OF THE LABYRINTH OPERATION.

According to my experience the labyrinth operation is in general not dangerous; especially is the opening of the vestibular apparatus without danger.

The technic is simple. Danger would only result from penetration of the inner vestibular wall and from injury to the dura in the inner auditory meatus. But this injury is exceedingly easy to avoid when the operation is performed *lege artis,* and when there are present no abnormal conditions such as are seen in the ossification of the labyrinth. Moreover, a highly situated jugular bulb can scarcely signify an appreciable increase to danger of life. Indeed, in consequence of narrow-spaced conditions, a small mastoid process, strong prominence of the lateral sinus, proximity of the cerebellar dura may render the operation difficult to such a high degree that the correct procedure is only with great precaution possible without injury to the adjacent organs.

Moreover, the opening of the cochlea performed *lege artis* appears to be unattended by danger, but having practiced the opening of the cochlea in twenty three cases only, while the opening of the vestibule has been performed by me over one hundred times, I should not permit myself to make a positive statement upon this question.

It seems certain, however, that in the opening of the cochlea injuries to the adjoining structures, *i. e.*, the dura in the inner auditory canal, occur much more readily, and may be regarded less as technical faults than in the opening of the vestibule.

Further, we have come to the conclusion that infection of the vestibule renders surgical interference necessary much more frequently than infection of the cochlea.

The complications of the deep extra-dural abscess, of empyema of the saccus endolymphaticus, and of the cerebellar abscess, occur only by this localization. The infection invades the vestibular apparatus through the oval window or the horizontal canal. The infectious vestibular disease is much more frequent than the cochlear malady and more extended and more progressive. The cochlear affection when fatal seems to have a slower course. For all these reasons the vestibular apparatus must come first in our consideration of operative treatment.

It will now be readily admitted that disease of the entire labyrinth represents a much more serious malady and renders the possibility of extension to the meninges decidedly easier than when only a single portion of the labyrinth is infected.

A warning to us are the three cases in which, after a successful vestibular operation, death was produced by meningitis, granulations being scraped out of the vestibule a long time after the operation, or, concurrently with this scraping out, Thiersch's skin transplantation being performed.

These cases demonstrate to us that perfect freedom from danger is not guaranteed even upon the evidence of the safety of the operation; danger to life may still arise from the subsequent treatment.

These dangers, however, diminish if they are anticipated, and, accordingly, the effecting of Thiersch's skin transplantation, as well as the accomplishing of the curetting, must proceed with the greatest care, or may be avoided entirely.

What indications are to be derived from our observations?

If we appreciate the freedom from danger of the opening of the vestibular portion and consider the dangers resulting from subsequent treatment as avoidable, we should be able to set up the principle, as soon as labyrinth disease has been diagnosed in one of the principal parts, that the operation is admissible in every case in the face of the dangers which may arise to the patient from the labyrinthine disease.

I have to state here that in this time of observation I have seen ten fatal cases among 190 cases of decided labyrinthine disease in which only the middle ear was operated upon. The labyrinth operation was naturally done in the most severe cases.

Now we have indeed acquired evidence, from a great number of positively diagnosed cases of disease, at least of the vestibule portion, that in a great number of cases the labyrinthine disease does not end fatally, nor in a chronic ailment, but effects a spontaneous cure.

In my opinion there is not the slightest doubt that in the majority of these cases disease of the entire vestibular portion has been present, even though single or numerous instances of it may be regarded as circumscribed disease of the semi-circular canal. We often found, a long time after the cure, negative caloric reaction.

We see from this, without doubt, that the labyrinthine disease, in many cases, ends in spontaneous cure, also that it does not always, in fact not once in the majority, incline to progression, but the cure resulting slowly and labyrinthine symptoms often lasting a long time.

The labyrinth operation is permissible in every diffuse infectious disease of the entire labyrinth or of one of its two portions, especially of the vestibule.

The operation should not be performed when it is a question of circumscribed disease in the labyrinth wall or in one of the semi-circular canals. As a rule this applies to the horizontal semi-circular canal.

When can circumscribed semi-circular canal disease be diagnosed and consequently labyrinthine operation be omitted?

1. When the labyrinthine symptoms are not present in excess.
2. When the function of the cochlea is not destroyed.
3. When the caloric reaction is violent, or at least well present.
4. When the fenestra ovalis is intact.
5. When acute middle ear suppuration is wanting, because of the tendency to diffuse labyrinthitis.

Carious processes on the outside of the labyrinthine capsule cannot be a reason for performing the opening of the labyrinth.

Operation is strongly indicated:

I. Where serious disease of the entire labyrinth or one of its two parts is present, usually the vestibule being affected.

II. When in stapes luxation after twenty-four hours, the symptoms given by increasing nystagmus, equilibrium disturbances and coated tongue grow worse. If fever appear, it is then highest time.

III. When the labyrinthine disease is complicated with infectious processes in the posterior cranial fossa, seldom in the middle

one; meningitis serosa, cerebellar abscess; caries necrota of the pyramid.

When is the malady of the labyrinth or only of the vestibule severe?

1. In acute middle ear suppuration every vestibular disease is serious and dangerous, and every fistula in the semi-circular canal is to be regarded as a sign of a present or imminent malady of the entire vestibular apparatus.

2. (a) Upon acute appearance of severe labyrinthine symptoms.

(b) Especially in severe labyrinthine symptoms, not arising from pressure of cholesteatomata, granulations, pus on the perforated horizontal semi-circular canal, the caloric reaction lacking.

3. When, under observation, the caloric reaction is lacking or diminishing, the function of the cochlea diminishes, the vestibular symptoms become gradually worse.

4. Fever without other causes.

5. In cerebellar complications in the posterior cranial fossa, especially meningitis serosa.

6. Severe labyrinthine disease is further recognizable during the operation by:

(a) Appearance of pus, granulations from the fenestra ovalis or another fistula.

(b) Greater necrosis, especially at the vestibular wall.

Caloric reaction will be lacking or decreasing except in 1 and 2, where it may be reduced.

Its lack, however, is not necessary, since it alone does not prove the severe malady.

I must especially adhere to the indication for operating in severe labyrinthine symptoms, when not being produced from the middle ear.

Only when they have existed for a long period and the vestibular reaction is well maintained, as well as the cochlear function, can one expect the result of the radical operation. I lost a few patients because, in spite of strong labyrinthine symptoms, I could not make up my mind to operate at once.

METHODS OF OPERATION.

In opening the vestibule I have for fifteen years employed two methods, which I made public in various lectures in the years

1893 1900. One method attains the exposure of the vestibule, inside the temporal bone, by leading along the horizontal semi-circular canal; the other by removing the posterior wall of the petrous bone from the posterior cranial fossa at the upper crus from the lower, and at the posterior crus from the upper cranial, as well as at the posterior crus from the horizontal semi-circular canal. Injury to two organs must be avoided—to the facialis in front, which may happen in the first method; to the dura behind, which may occur in the second method.

With a high position of the jugular bulb and the fossa jugularis, a lesion of the bulbus is also possible, and if the fossa jugularis stretches very high up into the temporal bone the bulbus may, especially when the opening is made from behind, be very much in the way of the operation, even making it impossible. A lesion of the inner wall of the vestibule with injury to the dura of the internal auditory canal, is almost impossible, if the operation is properly performed. But caution is necessary, the wall in part being very thin. The injury to the dura in the meatus would have fatal results. We once saw death ensue. The opening of the internal auditory meatus is most easily possible, if one loses the direction and penetrates above or behind the vestibule into the petrous bone, instead of forwards and downwards into the vestibule.

THE OPENING ALONG THE HORIZONTAL SEMI-CIRCULAR CANAL. SEMI-CIRCULAR CANAL OR TYMPANAL METHOD.

I usually begin the operation by removing the upper half of the horizontal semi-circular canal, and thus fix upon the anterior crus of the horizontal canal the limit below which one may not go. By operating carefully it is always possible to preserve the lower wall of the anterior crus; then the facial nerve is protected from lesion. At the posterior crus I remove the whole canal and so keep away from the roof of the vestibule. I also remove the bone under this part of the horizontal canal in the direction sloping down to the back, taking the very greatest precautions with regard to the facialis, which lies in front.

The stapes plate reaches with its posterior margin about the lateral boundary of the vestibule and takes up a middle position between horizontal and vertical. By this and by the extension of the ampullar extremities of the horizontal and upper canal, the upper portion of the vestibule leads toward the front.

If I penetrate in this way by the anterior crus in the direction of the canal, and above its lower wall towards the vestibule, and by the posterior crus close to its posterior and lower edge, I have selected a large portion of the lateral wall of the vestibule as the place of attack.

Thus it is easy to open the vestibule either in its posterior lower part, or near the ampulla of the horizontal semi-circular canal. According to the pathological changes the vestibule is to be opened here or there, in front, above, or at the posterior orifice of the semi-circular canal; in cases without fistuke, in the described way, along the horizontal semi-circular canal, in the direction of the oval window, parallel with, behind and a little below the facial bridge. Thus the facialis is surely protected from lesion.

The direction in which we have to seek the anterior wall of the vestibule is indicated, not only by the anterior crus of the horizontal semi-circular canal, which might lead us astray by its long extended position, but also by the fenestra ovalis, which we can in any case precisely locate by the introduction of a hooked probe. In cases of narrowing of the vestibule by new formation of bone we must expose the front part, at the same time preserving the portion of bone which supports the lower wall of the anterior crus, as a bridge. We penetrate behind and parallel with this bony ridge, inwards, downwards, forwards, in the direction of the fenestra ovalis. If the vestibule is found, the examination with a hooked probe will give a certain opinion as to the size, form and position of the cavity. The lateral wall may easily be felt with the probe, and then neatly removed with a narrow chisel or burr.

If one wishes to obtain information of the contents of the vestibular apparatus, the chisel is to be preferred. To prevent the anterior crus from snapping off, the burr is the better instrument.

By using necessary caution and by experience the facialis need not be exposed.

It is of the greatest importance to use the chisel as a plane, and to plane off shaving after shaving.

It is of importance to reach the deepest point of the vestibule, to lay bare its floor, to smooth the way to it properly, and to arrange that it may be seen at a glance. By exposing the lower orifice of the inferior semi-circular canal we enlarge the cavity of the vestibule downwards and by exposing the common orifice of the vertical semi-circular canals we enlarge it at the back. It is

precisely for the enlargement of the vestibule towards the back, either the better to reach diseased bone lying behind the labyrinth, or to create below more favorable conditions for wounds, either with or without exposure of the dura, that the burr is eminently suited.

Finally I exposed the ampulla from the upper vertical semi-circular canal, and then consider the operation completed. Only very rarely do I follow up all the canals, opening them in their full length, starting from the cavity of the wound.

The disease in the canals is usually slight, and after exposure of the orifices into the vestibule, the conditions for healing are specially favorable in them.

It is not necessary to expose it further from the fenestra ovalis. Only if the edge of the fenestra ovalis itself is diseased, or the wall of the labyrinth affected, and an operation on the cochlea becomes necessary, do we consider it expedient to scrape and enlarge the fenestra ovalis with the burr.

In general I warn against this, especially against vigorously enlarging the fenestra ovalis towards the back and below, for the bridge which supports the facialis is thus made narrower and necrosis is facilitated.

As a rule I employ this semi-circular canal method. In fact, I consider it the obvious method, in the ordinary forms of labyrinth disease, pointing to the safest way. Any danger to life is out of the question, is impossible.

I should most emphatically reject all attempts to arrive at an opening of the vestibule from the fenestra ovalis only. The widening of the passage is too inconsiderable, and can, in my opinion, not be of sufficient use, even if, by widening downwards to the back, the opening can be enlarged to the floor of the vestibule.

The cases which are cured by this method of operation would in all probability have healed quite spontaneously.

ENDOCRANIAL METHOD.

I have much more rarely employed the second method of exposing the vestibular apparatus after removing the posterior wall of the petrous bone and widely opening the posterior, and, when necessary, the middle cranial fossa.

I performed this operation for the first time in the year 1895, in the case of a deep extra-dural abscess which had penetrated into

the labyrinth. In the same year I also indicated the circumstances under which I considered it right to employ this method. In Blau's Encyclopedia I have fully described both methods.

Upon the radical operation follows next the removal of the posterior wall of the pyramid as far as the labyrinth wall. Starting from the back, the labyrinth is now removed as far as the middle of the horizontal semi-circular canal.

One begins by chiseling away the lower vertical semi-circular canal, the posterior crus of the upper one and adds to the removal of the posterior half of the horizontal one. The exposure of the posterior part of the vestibule is easy and without danger. Working at the dura of the cerebellum at this great depth is not quite so free from danger on account of the fixation of the dura here to the aqueductus vestibuli. The dura occasionally tears, fluid runs off, and even if the cases mostly take a favorable turn, one cannot be certain of a good result. I have seen such cases end fatally.

In order to expose the anterior portion of the vestibule thoroughly, one must remove some bone behind the anterior crus of the horizontal semi-circular canal and parallel to it.

The anatomical conditions at the temporal bone are sometimes very complicated, and abnormal position and development of single parts may render the carrying out of this method simply impossible.

The disease of the bone, in rare cases, goes deeper still, so as to compel one to penetrate at the posterior pyramid wall as far as the porus acusticus internus. It seems to me inadmissible to lay down this as a rule, although it has been proposed. We have to remove that which is necessary, not that which is possible. This endocranial method takes place in all complications in the posterior cranial fossa, and in all diseases of the bone which reach as far as the posterior pyramid plane (floor).

Here, also, the horizontal semi-circular canal, so easily recognizable, serves best as a means of direction and guidance.

It is desirable, also, to expose the anterior portion of the vestibule in such a way that from the auditory canal a sufficient view may be obtained and the treatment thus carried out. The small portions of bone which impede the view from the side and front can be removed without danger to the facialis.

Besides the entire removal of all diseased matter from the portion of the vestibule, this method has the advantage of being absolutely safe for the facialis.

If the exposure of the front portion of the vestibule is not carried out exactly from the back as here described, the enlargement of the entrance of the fenestra ovalis downwards and backwards is indicated.

This method is recommended by Neumann as the standard operation in the opening of the labyrinth, and is constantly performed. I cannot agree to this plan, for the operation is certainly rendered more difficult by the extensive exposure of the cerebellar dura, and involves the danger of perforation of the dura and imminent meningitis.

The quicker healing is of itself no sufficient reason for the regular employment of this method. Healing would take place even more quickly if the whole of the upper wall of the pyramid were removed.

The sinking of the temporal dura at once causes a quite considerable contraction of the cavity of the operation, but this contraction does not always mean an acceleration of the healing process. It may very frequently have an extremely detrimental effect.

I endeavor to avoid exposure of the dura, if possible, and consider the preservation of the bony edge of the mastoid an advantage well purchased under certain circumstances, even by a somewhat slowed healing process. Still, I by no means wish to express the opinion that in this way the healing is in general delayed. Nor, consequently, for accelerated healing (cure) do you perform every radical operation, also removing the posterior and upper petrous wall.

The semi-circular canal method is, in my judgment, very much simpler and easier. Pathological changes, the construction of the temporal bone, may be such that this method is by far the easiest.

In a serious destruction of the horizontal semi-circular canal, where, for the complete opening of the vestibule, it is perhaps only necessary to remove 1 or 2 mm. of bony substance, no one will propose the far more dangerous and troublesome, and also technically difficult, opening from behind, and if the facialis should have to be entirely destroyed, there is certainly no reason for considering this method the standard one.

But there is yet another objection which I should like to raise at this point, and that is as to the name which has lately been given to this method of operation.. Ten years ago it was known as my

method, later it was called the Neumann-Jansen method, and now is only known in Vienna as Neumann's method.

Although it is a great honor to me that my method should have been adopted by so distinguished a surgeon, and believing that the opening of the labyrinth is not a difficult feat for another to have done, still I consider it my duty to say that I wish to keep the share that I have had in the invention of this method.

OPERATION UPON THE COCHLEA.

Although formerly I proposed the complete removal of the cochlea, and have often performed it, in recent times I have restricted myself to the removal of the promontory and the exposure of the lower cochlear whorl for the preservation of the modiolus. Having seen one case end fatally in which, after complete removal of the cochlea, the dura in the interior auditory canal was perforated by probing, and discharge of the fluid lasted several days, I have grown cautious.

In order to obviate this with safety, it is necessary to limit oneself to the removal of the promontory, to the exposure of the lower cochlear whorl, without injuring the modiolus itself. Should one inadvertently or, owing to destruction in the cochlea, arrive at the bottom of the inner auditory meatus, the probing in this dangerous region, and the opening of the dura, must be avoided. Upon the opening of the subarachnoidal space the cerebro-spinal fluid appears, mostly to a considerable extent and of several days' duration.

In order to guard against any possibility of lesion to the subarachnoidal space it seems expedient not to interfere with the modiolus at its base, but to leave it here undisturbed. With these propositions of Richards I entirely concur.

When the cochlea in the interior has become transformed into a bed of sequestra, pus and granulations, the removal can be effected without danger by means of the gouge. I generally perform the operation either with a chisel or a burr, the smallest chisel for preference, beginning with the enlargement of the fenestra ovalis towards the front below. If the burr is used, the mode of operation is very simple; it is introduced into the fenestra ovalis, the bone is chipped off towards the front below at the promontory, corresponding to the lower cochlear whorl.

Dr. Wendell C. Phillips moved that a vote of thanks be extended to Dr. Jansen for his presence at the meeting, and for the masterly oration just delivered. Seconded and carried by a unanmous rising vote.

DISCUSSION.

Dr. Edward B. Dench, of New York City, in opening the discussion, expressed for the Society the deep appreciation felt for the honor conferred by Dr. Jansen in being present, and for the masterful exposition of the subject of infective labyrinthitis. Dr. Jansen was to be congratulated for his wonderful work, and for the interesting and extensive series of cases presented. Dr. Dench had operated twelve times for labyrinthine suppuration. The cases were not as severe as those reported by Dr. Jansen. In some of them the labyrinthine suppuration was found at the time of operation, careful curettage of the area of the fistula showing the disease. It had been his practice to curet, as suggested by Dr. Jansen. The point was well taken that the smaller the canal the less the danger of extension of the disease; consequently, in a case with no symptoms and with simply a gross lesion, if the disease were removed it was not necessary to proceed to drain the labyrinth. In his cases the disease was suspected on account of vertigo or vertigo and nystagmus. He had followed Dr. Jansen's plan of conservatism, but, contrary to his advice, he did enlarge the fenestra ovalis. He had no facial paralysis, but in one or two cases there was slight paresis. These cases were followed by cure. In one case he found it necessary to remove the cochlea. He was convinced that labyrinthine symptoms are present in many cases immediately after radical operation, or in cases of suppuration of the middle ear, and he believed the reason American observers had not seen them more often was because they had not looked for them. This bore out Dr. Jansen's contention that a certain number of cases in which there was a simple serous effusion in the labyrinth would get well without radical operation. Dr. Dench emphasized the importance of making delicate manipulations about the oval window, and of being ready to enlarge the oval window and to drain the labyrinth through it should the symptoms indicate such procedure.

Dr. Canfield, of the University of Michigan, said that he had listened with the greatest interest and benefit to Dr. Jansen's paper, and felt that we could congratulate ourselves upon having had the

opportunity of hearing this masterly discourse upon a subject of such importance and from an authority so able by virtue of years of experience and a remarkable operative skill to decide wisely the questions involved. Having been intimately associated with Dr. Jansen in the past, it was not remarkable for him to do himself the honor to agree with him in many of the points that he had made.

Those symptoms generally considered diagnostic of labyrinth involvement, i. e., nausea, vomiting, vertigo, disturbance in equilibrium, and nystagmus are noticed usually only when the labyrinth has been suddenly invaded, or during extension of disease within a labyrinth already involved. He said some authors maintained that von Stein's sign was present at all times, and in all cases of labyrinth suppuration. In one case, at least, he had been unable to note any evidence of its presence, although a fistula in the semicircular canal was demonstrated at the time of operation. He believed there was a tendency to exaggerate the importance of the swaying movements made by an awkward person or by a sick one while trying to maintain the erect position after going through such gymnastics. Again, as von Stein's sign is an indication of disturbed labyrinth function, but not necessarily an evidence of infection, it may be present during the course of a chronic suppurative otitis media without actual erosion of the labyrinthine capsule. He thought we all had noted marked disturbances of equilibrium in patients with chronic suppurative otitis media in whom no fistula into the labyrinth could be discovered, and in whom all symptoms disappeared after the radical operation.

He wished to emphasize the fact that marked deafness, while an important symptom when present, was not present in all cases of involvement of the labyrinth. When a patient suffering from a chronic suppurative otitis media developed under observation a sudden marked increase in deafness in the suspected ear the symptom, he thought, was of great significance. The absence of marked deafness did not necessarily mean, however, that extensive pathological change had not already taken place in the labyrinth, for not only might the vestibule be filled with pus, but the cochlea might also be invaded without destroying hearing for the conversational voice. In his own cases, the hearing for the voice had varied from a whisper at three feet to a degree that made it probable that the loud conversational voice was heard in the other ear, although that

ear was hermetically sealed. The fact that a very high degree of deafness often exists without extension of the infection to the labyrinth, and that extension of the disease to the labyrinth sometimes takes place without causing marked deafness, lowers the value of the voice test, he thought, considerably. The Weber was decidedly unreliable. In several of his own cases it was not localized quite as often in the suspected ear as in the other. The Rinné was negative more often than it was positive. The high limit was always lowered.

He said the diagnosis of labyrinth suppuration was at present made with certainty, in the majority of cases, at least, only at the time of the radical operation by the discovery of disease of the inner mastoid or tympanic wall.

He thought much difference of opinion existed concerning the pathological significance of perforations in various portions of the internal labyrinth wall. In his own cases, judging from hearing test and result of operation, single perforations in the horizontal semi-circular canal occurred only in cases of circumscribed labyrinth disease. Double perforations, one in the horizontal and one in the posterior semi-circular canal, occurred in two cases in which the vestibule was filled with cholesteatoma and pus. Double perforations, one in the horizontal semi-circular canal and the other through the oval window, occurred in extensive involvement of the labyrinth. It would seem that double or multiple perforations must always speak for an extensive pathological condition. In considering the value of these perforations, he felt that it should be considered that disease had been demonstrated clinically at autopsy in cases in which perforation could not be recognized.

The indications for operation upon the labyrinth, in his opinion, were met in two classes of cases:

1. Those in which symptoms referable to labyrinth disease had not been present before operation.

(a) When a single perforation was discovered through a semi-circular canal, from which pus escaped.

(b) When a perforation through the oval window was discovered, whether pus was seen or not. In these cases, marked deafness and extensive disease of the vestibule must be suspected.

(c) When labyrinth symptoms developed suddenly after a radical mastoid operation. The majority of these cases were due to a latent labyrinth suppuration, an acute exacerbation of which had

been set up by the operation. In such cases, the vestibule was to be opened widely, and further operative measure governed by its condition. In isolated perforations through the horizontal semi-circular canal in which no other evidence of disease could be demonstrated, operation upon the labyrinth might be postponed. This was in support of Hinsberg's statement that, in his experience of all cases of circumscribed semi-circular canal diseases, not a single patient succumbed to the consequence of the labyrinth suppuration.

In this connection, he pointed out the necessity of watching very carefully cases in which a single perforation of a semi-circular canal had been demonstrated, so that on the appearance of vertigo, nystagmus and diminution of hearing, the labyrinth may be promptly opened.

2. Those in which labyrinth symptoms had been present before operation.

(a.) When no fistula was found.

(b.) When fistula was found.

When no fistula was found, this class of cases, it seemed to him, should be very small and should include those cases which have developed labyrinth symptoms while under observation and those in which deafness typical of disease of the perceptive apparatus could be demonstrated. He thought symptoms highly suggestive of labyrinth suppuration so frequently disappeared after the radical mastoid operation that unless the pathological change in the tympanum and mastoid was altogether too slight to account for the labyrinth symptoms, delay might be allowed. To this class belonged also those cases in which a deep-seated cerebellar abscess or extra dural abscess was suspected (Jansen).

(a) When a perforation into the labyrinth was found in a case that had previously shown labyrinth symptoms, the indication for opening the labyrinth was clear.

In all cases in which labyrinth suppuration had been suspected before operation or had been diagnosed at the time of operation by the discovery of a fistula leading into the labyrinth, the radical mastoid operation should, of course, be performed. The last steps in this operation included, he thought, as careful and thorough delineation as possible of the anatomical structures in the inner wall of the mastoid and middle ear. Only by the most painstaking investigation of this wall, could an intelligent decision be reached as to the condition of the interior of the labyrinth. The

method of operative attack upon the labyrinth might vary with the location of the fistula. If the fistula was discovered through some part of the semicircular canal system that portion of the labyrinth was entered by uncovering the canals widely.

Dr. Canfield went on to say that the chief reasons for entering the labyrinth through the canal system were that it was more easily accomplished, was attended by less danger to life than was an attack through the oval window, was sometimes followed by less damage to the hearing and finally sometimes revealed a localization of the pathological process that makes further operative interference unnecessary. During this step, he made it a practice to uncover the facial nerve for some distance in its horizontal portion in order to learn its course perfectly and thus to avoid injuring it. The operation upon the vestibule, through the oval window, might follow the opening of the vestibule through the canal system if the extent of the pathological process warrants it, or it might be undertaken first if a sinus through the oval window was noted. It was attended by greater danger to the nerve and to life by the immediate proximity of the inner wall of the vestibule, fracture of which was to be considered of the greatest danger. The operation upon the cochlea, it seemed to him, should be made only if definite evidence of involvement of this portion could be seen. This step was by far the most dangerous part of the entire operation, not only on account of the most intimate relationship between the carotid artery, the jugular bulb and the facial nerve, but also on account of the position of the hollow modiolus which occupies the center of the cochlea. On account of the fact that the internal auditory meatus terminates between 1-8 in. and 3-16 in. internal to the promontory, to do more than uncap the pyramid must be exceedingly dangerous. Personally, he preferred to secure a good view of the upper whorl and a fair view of the second by removing the promontory and uncapping the pyramid and stopping there, even though a portion of the lower whorl had not been seen. Of course, if the entire cochlea was loose, it might be removed. Even if caries could be demonstrated in the lower whorl, it seemed to him safer to allow this portion to sequester rather than to remove it with the probability of opening the internal auditory meatus.

Dr. Kaspar Pischel, of San Francisco, Cal., asked Dr. Jansen if he had found the use of antistreptococcic serum advantageous as a preventive measure. He referred to the custom of Dr. Cushing, of Baltimore, of using urotropin in all cases of brain operations

because in a few hours after taking urotropin, formalin appears in the spinal fluid.

Dr. Robert C. Myles, of New York City, had had three unique cases of labyrinthine disease. One was caused by an injury to the semicircular canal during operation, and the phenomenon of this nystagmus and general disturbance of lachrymation existed in addition to which the patient had to lie down nearly all the time for a week or two, and when there was a sudden noise she would be thrown into a tremor.

The patient ultimately recovered. Another patient had an epithelioma in the zygomatic region, to which an escharotic was applied. In some unknown way, the destructive agent went into the external auditory canal, where everything was completely destroyed but the cartilage. The question arose whether complete operation should be attempted or whether nature should be allowed to take care of the process. He made a slit in the external auditory canal, removed the external osseous wall and the annulus tympanicus, and when the labyrinth was reached, it was found to be practically dead. He removed the greater part of the semicircular canal and cochlea, and the patient made a complete recovery, with perfect hearing, remaining well to the present time. This man's son, nine years of age, had a discharge from the cochlea. Dr. Myles removed what was left of the membrana tympani, also the ossicles and curetted quite a distance into the cavity. These cases were illustrations of the fact that labyrinthitis, if looked after, is not such a desperate condition as it is sometimes considered.

Dr. Thomas J. Harris, of New York City, asked Dr. Jansen, in closing the discussion, to give his opinion with reference to a group of cases in which labyrinthine symptoms accompany acute affections of the middle ear. In America, otologists were of the opinion that such symptoms disappear after proper attention to the middle ear and mastoid, and that consequently a conservative course was all that need be followed.

Professor Jansen, in closing the discussion, said: In answer to Dr. Richards' question, I would like to say that I consider the caloric reaction of Barany to be very valuable. Through it, the latent disease is made manifest, which is undoubtedly a decided advantage. But, as an indication for operation on the labyrinth, the caloric reaction has not the same significance.

If Dr. Richards thinks that Neumann's operation is different from that described by myself (regularly to open the vestibule and cochlea, and to incise the dura), I would like to answer, that about fifteen years ago I made the suggestion, to open the vestibule and cochlea widely, where they are diseased, and there is indication for operation. I have often described both the operation and the methods. In the course of years, I have learned to restrict my operation to a portion of the labyrinth, and to individualize in my operations, whereas Neumann always opens the entire labyrinth. Even though he has made a few unimportant modifications upon my operation, the operation can hardly be called his own. The next few years will decide whether my present standpoint is the proper one, namely, to individualize, or Dr. Neumann's standpoint, to generalize, and open the entire labyrinth. I do not consider it indicated to make an incision in the dura in every case.

In reply to Dr. Harris, I would like to say that I have often observed cases with labyrinthine symptoms before the radical operation heal, without an operation on the labyrinth. In the majority of cases, changes in the middle ear probably caused a reaction in the vestibular region. I do not operate on every case with labyrinthine symptoms. I have laid particular stress upon the infectious and progressive character of the labyrinthine affection. On account of a too conservative attitude, and too late an operative interference, I lost one case. It is very difficult to draw the line between the cases which ought not, and the cases which ought to be operated upon.

I have used antistreptococcus serum, systematically, as a prophylactic agent, in a series of cases, but without result. The dose was 10.0 gm.

PAPER:

THE POST-OPERATIVE EFFECTS OF THE STACKE OPERATION.

JAMES F. McKERNON, M. D.

The programme gives the title of this paper as the post-operative effects of the Schwartze Stacke operation, but this is incorrect, as I shall speak only of the effects of the Stacke operation.

When a patient consults us for the best methods of curing a running ear that has persisted for a long time, there are many points which we, as otologists, should consider. First of all, is it necessary that a radical should be done? If dead bone be present in the tympanic cavity the answer is in the affirmative, but there are many cases where the patient is advised to have this operation performed on account of a discharge from the middle ear, which has been in existence only a few weeks or months, and where no dead bone can be demonstrated. In this class of cases I believe it is a very unwise procedure. I do not wish to be understood as opposed to this operation, when there are indications distinct and definite for its performance, for when these are present, I do not know of any other method of treatment that offers the patient such complete relief and safety. From personal observation, however, I believe it is frequently advised and performed where there is absence of necrosis, and before other measures of a more conservative nature, in the way of local treatment, have been exhausted. In discussing the post-operative effects, the question of the percentage of recurrences is an important one. It is my belief that fully one quarter of the total number operated upon lapse into the recurrent class, and yet many operators will tell us that the number of their recurrences amounts to only four or five per cent, and they are right in believing this, for a large number of these cases operated upon from clinical material in the hospital pass out from their immediate observation in from two to four weeks, and drift to other hospitals, where they again apply for

treatment for a discharging ear. The cause of these recurrences is not always failure to remove all the dead bone, though frequently such is the case, but many of them are the result of faulty technique in dressing after they have been discharged from the hospital wards. It is just as important for the cure of the patient that he who dresses the case after operation, should have had experience in this special line of dressings, as it is that the operator should have had experience in his field, and many an operator would have more complete cures to his credit, were he to follow up and dress his cases personally. Failure to properly rim out and remove all diseased tissue at the tympanic orifice of the Eustachian tube is also the cause of a certain number of these recurrences. But a still further cause acting by the way of the Eustachian tube must be considered, and that is, that many of these patients contract colds easily, the tubes become infected, and this extends to a mass of new scar tissue lying over the tube with the result that it becomes broken down, and a direct communication is once more established between the tympanic cavity and pharynx. The cause for such an extension in a large number of the cases is due to the mechanical obstruction in the naso-pharynx by a mass of adenoid tissue and frequently this is markedly aggravated by a chronic amygdalitis. I have seen several cases recur, when upon the second operation there was not a vestige of diseased bone in the middle ear cavity, all the trouble being at the lower end of the tube.

The effect of the operation upon audition is one of, if not the most, important to the patient, and I believe it is not discussed as frankly with the patient about to have this operation, as it should be, for many have volunteered the statement, that, had they known their hearing would be so poor, they would never have had the operation done. I have never seen but one case where the hearing was improved and remained so following operation, nor do I believe in the majority of cases that an improved permanent audition is ever the result of that operation. To be sure, there are a number of cases reported with most brilliant results in regard to audition, but the reason for this is, no dead bone was present when they were operated upon, and in all probability recovery would have taken place by a more conservative method of treatment, and a greater amount of audition been spared to the patient. In the vast majority of cases at the end of six months or a year, the hearing for all practical purposes, principally that of conversation, is a thing of the past and never returns.

Tinnitus. In a certain number of cases operated upon the tinnitus is improved temporarily, but after a substantial mass of scar tissue has re-formed, it again becomes troublesome, though not as a rule to such an extent as before.

Pain. The pain in nearly every case is relieved after operation.

Vertigo. That which has been caused by pressure and not by labyrinthine disease is invariably relieved, and even though the patient exhibits marked vertigo prior to operation, the labyrinth should not be molested, unless we find a necrotic process invading it.

Nausea and vomiting are relieved in the majority of cases, as well as unsteadiness of gait, unless the labyrinth is involved.

When mental symptoms are present they are greatly relieved, and in the majority of cases give no further trouble, and there is a gradual improvement in the whole system.

Facial Paralysis. This occurs in a number of cases operated upon, but fortunately it is in the majority of cases but transient. It is wise, however, to acquaint a patient about to undergo this operation of the possibility of such an accident taking place. Should the nerve fail to perform its usual function at the end of a month after the operation, the use of an interrupted galvanic current will hasten resolution very materially.

Among the many reasons given why the radical operation should be performed is, that if the disease be allowed to proceed unchecked, there will eventually be an involvement of the opposite ear, non-suppurative in character, or of the nerve. I have seen a few cases of long standing to which no other attributed cause could be assigned to this bilateral invasion. How frequently this occurs, I am unable to say, but think it is oftener than was formerly realized.

The cosmetic results of a Stacke pure and simple are always good, barring accident, such as an infection of the suture line or a perichondritis of the auricle. In fact, it would require close scrutiny to detect a scar in the majority of cases one or two years after the operation was done, and the meatal opening, though larger than normal, is by no means disfiguring. A question which most patients ask prior to operation is, "how long must they be incapacitated from their usual daily routine?" This varies with the individual, much depending upon the general condition at the time of operation. In an average case, they can be up and around at the end of a week, and can, in a measure, resume their usual

duties in from four days to a week later, making the time limit they are away from business two weeks. In the average case there is complete dermatization in from six to seven weeks from the time of the operation. A few cases heal completely in from four to five weeks, but this is by no means the average, and is independent of either a primary or secondary graft. In a suitable case the placing of a secondary graft on the operative field does, in a few instances hasten rapid dermatization, but it has so seldom been successful in my hands that I have practically abandoned it. In advising this operation for our patients, a most important question should be considered, that of mortality, and it is impossible to gather statistics upon this point, for operators are prone to report successes and not the failures.

That deaths do occur at times following this operation, we who are doing this work all know, and I believe that every one doing a considerable number of these operations could report, if he would, a certain percentage of deaths. If this be true, and I believe it is, should we not then be a little more guarded in our prognosis when the operation is under discussion with the patients or their families? I have had two cases of meningitis, and two of thrombosis of the sigmoid, bulb and vein, following, I believe, as the direct result of this operation, and all were fatal. I have also seen ten cases of meningitis, in consultation, following the operation, all of which were fatal, and six cases of sinus involvement and two of brain abscess of the acute variety. Three of the six sinus cases recovered after evacuation of the sinus and resection of the vein, one of the abscess cases recovered, the other died. I do not wish by the report of these fatalities to be understood as condemning the operation, for I am unqualifiedly in favor of it, when the local conditions and symptoms call for surgical relief, but I do protest against advising this operation, without informing the patient of its gravity.

To state that it is a simple operation, is to say what none of us who are doing this work believe, for I do not know of any more delicate, difficult and tedious work in the whole domain of surgery, than to do this special work as it should be done, and it is for this reason that I believe that no beginner should for a moment think of obtaining his experience in this line first from his patients, but should do as many operations upon the cadaver as he can, as a preliminary step to perfecting himself in the technique of this particular work.

PAPER:

CHRONIC RECURRENT SUPPURATIVE OTITIS MEDIA AND ITS RELATION TO MASTOID AND INTRACRANIAL COMPLICATIONS.

S. MacCUEN SMITH, M. D.

Intermittent Otic Discharge More Dangerous Than the Constant Variety.

In considering the subject of chronic recurrent suppurative otitis media, I will endeavor to demonstrate that this particular form of aural disease is relatively productive of more complications, intracranial and otherwise, than the continuous variety. I have been particularly impressed with the importance of this subject from the fact that the belief still persists, both among the profession and the laity, that an aural discharge which is intermittent is of little importance, the assumption being, therefore, that the underlying disease is never dangerous to life. This fact clearly illustrates that the pathology and prognosis of these conditions are not generally understood, notwithstanding the marked progress which otology has made in this direction during the past decade.

Our Duty to the Profession and the Laity.

For years past many of us have been persistent and untiring in our efforts to impress upon the profession the importance of aural disease, and to indicate the obligation due our patients in the prompt recognition and treatment of the same. Like all weighty medical problems, the evolution has been slow, but we have at least established a fairly good general conception of the acute diseases, their dangerous complications and ofttimes tragic sequelae. As the general surgeon has persistently urged, year after year, the necessity of early operative interference in appendicitis, so must we labor to secure a proper realization of the actual dangers of a discharging ear. Furthermore, we must not only recognize this

condition, but must properly appreciate its gravity and demand the adoption of prompt and efficient measures for its relief.

This widespread indifference to a very grave problem is the natural outcome of continued apathy on the part of the medical profession, and if the Fellows of this Society are to conserve her honorable traditions, it becomes the immediate and imperative duty of each and every one of us to spare no effort, however self-sacrificing, to place before the profession some plain facts relative to the ever-present dangers of an otorrhea. We must, therefore, accept the obligations and responsibility created by such circumstances, and resolve to diffuse, in the most effectual manner at our command, such information as will throw proper and responsive light on this subject before the great body of practicing physicians, who will, in turn, be moved to action, and will thereby become the legitimate instructors of their patients, the laity.

Value of Prophylaxis.

In this connection I wish again to take the opportunity of pointing out the great value of prophylaxis as applied to mastoid and intracranial involvement, complicating acute tympanic disease. In the primary stage we find a simple hyperemia and swelling of the mucosa, together with some sero-mucous exudate. At this particular period probably four-fifths of all cases will yield promptly if the well-known simple measures for their relief are immediately applied, whereas the vast majority of all serious complications are directly traceable to the unpleasant fact that these primary measures have been partially or wholly neglected.

I am not unmindful of certain cases complicating the exanthemata, influenza, etc., which seem to be virulent and purulent from their very inception, but even in such cases a free incision of the membrana tympani before bulging occurs will usually prevent serious involvement of the accessory cavities and adjacent structures. Such clearly defined facts should, therefore, convince us that a discharging ear, whether of the acute or chronic form, is frequently the result of woeful ignorance or indifference, and that the subsequent mastoid and intracranial complications are mainly chargeable to the same cause.

At times we are confronted with the statement that an otorrhea has existed for many years without producing untoward symptoms. So long, therefore, as the patient experiences no actual suffering

he is content, often by the advice of his physician, "to let well enough alone." This viewpoint is quite natural and excusable on the part of the patient, but it does seem inconceivable, in this day of progressive and preventive medicine, that those charged with and professing to conserve the public health, should so far forget their obligations or misinterpret their humane duty.

As a body, those practicing the learned profession of medicine are among the most honorable, and certainly the most self-sacrificing men to be found in any calling; consequently, this lackadaisical attitude toward aural disease that we too often meet with can only be attributed to misconception or misinformation as to the worth and importance of the subject. Such being the case, I again wish to proclaim this society, individually and collectively, the disseminators of such information or instruction as may be necessary for the betterment of our profession, which, when viewed in the light of civic duty, must be accepted as an imperative obligation due to our fellow man.

Illustrative Cases.

In order to properly illustrate the destruction that frequently accompanies a recurrent suppurative otitis media, I will briefly relate the histories of three typical cases, which I have recently operated upon.

Case I. History. Male, T. W. D., aged 44 years. There is some doubt as to whether he suffered from aural disease early in life. During the past three years he has had a recurrent suppurative otitis media of the left ear, the primary cause of which is unknown.

The patient came under my care at the Jefferson Medical College Hospital, April 3d, 1908, stating that for the past three or four days he had suffered very severe pain, not only in the ear and over the mastoid process, but also on that entire side of the head, so much so that hypodermic injections of morphine were necessary to relieve his suffering. The history furthermore states that these acute exacerbations occurred about every three or four months. In the interim the ear would be entirely dry and free from pain. Prior to last attack, however, he was free from all aural symptoms for about eight consecutive months.

The examination showed the external auditory canal to be filled with a copious, brownish-yellow, foul-smelling pus. There was

a good-sized carious opening through the posterior wall of the external auditory canal, and communicating with the mastoid antrum, through which pus was escaping. There was much swelling, redness and tenderness over the mastoid process, extending upward to the superior border of the temporal bone, anteriorly to the outer canthus of the eye and posteriorly to the occipital protuberance. Pressure on this swelling would force a large quantity of pus through the carious opening already mentioned into the external auditory canal. The posterior half of the membrana tympani was destroyed; the anterior portion, however, remained intact, and was quite normal in appearance.

Patient's temperature at this time registered 103°. Operation was advised and refused. The following day he returned, still suffering, the temperature rising to 104° at noon. Operative interference was strongly urged, but again refused. The next day he came again, at noon, his temperature at this time registering 105.2°. He was not able to secure sleep or relief from pain even under the influence of hypodermic medication. He now readily consented to an operation, and was admitted to the hospital for this purpose.

Operation. A free incision through the soft parts evacuated a large quantity of pus. This incision was carried from the tip of the mastoid to about one inch above the superior border of the auricle, and then forward to the outer canthus of the eye for a distance of about two inches. A transverse incision on a level with the mastoid antrum was carried almost to the occipital protuberance. This large field of operation was necessaary on account of the extensive osteomyelitis present. In this particular case the extension from the diploe, which is usually first involved in cases of osteomyelitis, was external rather than internal, probably taking the course of least resistance. The entire external plate of bone was very dark, in some spots almost black, with numerous points of hemic ooze. All this area of necrotic bone was removed. There was a large carious opening in the mastoid process a little below the level of the antrum. The remaining part of the process was also discolored and showed many bleeding points. The necrosis was so great that the whole process was removed, including the tip. The sinus, as so frequently occurs in these chronic cases, was situated rather superficially and far forward, so much so that part of the posterior auditory canal acted as the anterior bony

wall of the sinus. The necrotic process had exposed the entire sinus, which was bathed in pus, and covered with a thick layer of what seemed to be healthy granulation tissue, with the exception of one part. The granulation tissue was removed on account of some bleeding, and this bleeding was increased by the removal of this tissue, although no opening in the sinus could be found. The dura was largely exposed, both through the tympanic roof and the antrum. The discoloration of the dura through the antral opening was so marked that it suggested the possibility of some intradural abscess formation. A hypodermic needle was introduced for the purpose of diagnosis, but with negative results.

The septic temperature ranged from below normal to 106°, and continued for some days after the operation. The man at times became quite delirious, and the whole picture resembled one of septic sinus thrombosis. My own conviction, and the judgment of the various consultants, favored the opinion that the man was suffering from a general septicemia. Hence, no further operative interference was resorted to. The wisdom of this conservative decision was borne out by the fact that after the patient had passed through superficial multiple abscess formations in various parts of his body, he made a complete recovery.

Blood cultures and differential blood counts were made at various stages of this patient's illness. The former demonstrated the presence of the staphylococcus pyogenes albus, and the latter showed, as might be expected, a marked hyperleukocytosis and a decided increase of the polynuclear percentage. This relative ratio between the hyperleukocytosis and the polynuclear percentage continued throughout his illness, clearly demonstrating the value of the blood count in all septic conditions.

Frequent examinations of the eye-ground gave negative results.

One point of interest and importance to which I especially wish to direct your attention, is the fact that we can hardly conceive that all the bone destruction shown in this case could have occurred in the three or four days which marked the acute exacerbation of the chronic recurrent suppurative otitis media, and yet the patient and his family claimed that for eight months prior to the date of this acute exacerbation he did not suffer the slightest inconvenience from his ear and that the same remained entirely dry during that period.

This brings up for consideration the extensive pathologic changes that apparently take place without causing the patient any discomfort. We are frequently surprised at the extensive necrosis met with in operating for the relief of mastoid disease. This remark applies both to the acute and the chronic form of mastoid disease, but of course is much more common in the latter. From this experience, especially during epidemics of influenza, I am convinced that bone destruction does sometimes occur with little or no inconvenience, until a certain point has been reached. In other words, this furnishes us an additional argument in favor of comparatively early operative interference.

The fact that this patient was able to attend the dispensary service on three consecutive days, with a temperature ranging from 103° to 105°, would indicate that he possessed an unusually good constitution, and that he could not have had any serious intracranial involvement.

Case II. History. Another case which well illustrates the point under discussion is that of H. D., aged 18 years, who suffered from a recurrent suppurative otitis media of the right ear from early childhood, the trouble recurring at intervals ever since. At times the discharge would cease for a period of about one year, the membrana tympani completely regenerating in the interval, the pain, however, being much worse during the period of cessation of discharge. Whooping-cough is the only disease from which the patient ever suffered, and this is said not to have had any influence on the aural condition.

An acute exacerbation occurred about two years ago and was very severe, the discharge lasting until he applied for treatment at the Jefferson Medical College Hospital, October 11, 1907. Examination at that time revealed the presence of a slight discharge in the external auditory canal escaping from a small perforation in the antero-posterior part of the membrana tympani, the remainder of the membrane being intact and looking fairly healthy. The Eustachian tube was patulous, an increased amount of secretion being forced through the perforation by the use of Valsalva's method of inflation. Deep pressure over the mastoid process elicited some tenderness. In order to provide for better drainage, the membrana tympani was freely incised and the patient placed on the usual treatment.

November 23rd, 1907, or about five weeks later, the drumhead was found to have entirely regenerated and was quite normal in appearance, the patient, however, complaining of severe pain over the mastoid process and the right side of the head, although, with this exception, there was not the slightest evidence of the classical symptoms of mastoid disease, as evinced either by redness or swelling over the process or by any inflammatory condition of the drumhead. There was, however, some drooping and slight redness of the superior and posterior wall of the external auditory canal.

When the patient first applied for treatment, the mother was advised that an operation would probably be necessary. The condition had now improved to such an extent that we advised deferring operative interference. This did not meet with the approval of either the patient or his mother, the latter stating that the boy had been going through this process practically all his life, and she was convinced that the recurrence of pain on that side of his head would soon be followed by a reappearance of the discharge and that at such times he usually became quite ill, with high fever, severe headache, nausea, vomiting and great prostration.

Operation.—Heath's operation was performed on December 6, 1907. Here again the mastoid was found to be of the diploic variety, the accompanying osteomyelitis extending back almost to the occipital protuberance. This, of course, offered a ready explanation of the pain on pressure over the mastoid and posterior to the same. As in the former case, extensive necrosis was found, especially over the sinus, exposing the same for almost its entire extent.

The sutures were removed and the dressings changed six days after the operation. The wound was dressed every second day until January 2, 1908, when the patient was allowed to leave the hospital apparently well. On February 15, 1908, or about two months after the operation, the patient returned, the discharge having entirely ceased and the hearing having shown marked improvement. On April 17, or about four months after the operation, the patient again reported for examination. The membrana tympani was now entirely regenerated, and there was a complete cessation of the discharge. The hearing had improved almost to normal, and he was practically free from all discomfort.

The result, in so far as the hearing is concerned, shows the importance of resorting to a Heath or a modified radical operation in all suitable cases.

The destructive nature of a recurrent suppurative otitis media, to which little or no attention is usually paid, is well illustrated by a patient from the Far West on whom I recently operated.

Case III. History. Married woman, aged 38 years. Without apparent cause she has had a recurrent suppurative otitis media of the left ear practically all her life. Up to eight years ago the discharge would appear about every two months. At that time, however, it ceased until nine months ago, when the acute exacerbation was ushered in with characteristic headache, nausea and great lassitude, extending over a period of several days, until the discharge again appeared, which in turn relieved the pain considerably, as so often occurs. The otorrhea, however, had continued during this entire nine months. In June, 1907, she had an acute suppurative otitis media of the right ear, which was the first time this ear had been involved.

The patient consulted me April 7, 1908. She was found to be suffering severely with headache, confined to the left side. There was no swelling over the mastoid process, but she suffered from rather severe pain on gentle pressure over the mastoid, and extending for some distance posteriorly. There was a copious discharge escaping from the external auditory canal, of a brownish-yellow character, streaked with blood and very offensive. The entire membrana tympani in this ear was practically destroyed, as well as the ossicles, which, however, were said to have been removed on a former occasion. The superior and posterior wall of the external auditory canal was very red, extremely sensitive, and drooping. The hearing was practically nil.

Operation. In this patient we found the cortex exceedingly hard, it being flint-like in character. The sinus was pushed far forward and exposed by the necrotic involvement. There was some necrosis of the bony roof of the mastoid antrum, which, on being removed, evacuated considerable pus from an extradural abscess.

The radical operation was performed in this case. The patient made an uninterrupted but rather slow recovery, on account of suffering from an intercurrent attack of purpura hemorrhagica

with hemophilia, the bleeding taking place at many points over the entire surface of her body, but more especially in the throat and gums. Although no undue hemorrhage was met with at the time of the operation, there has been a tendency since that time to have some bleeding from the mastoid cavity. On the other hand, up to the present time there has been no recurrence of the purulent secretion. The vertigo from which she suffered prior to the operation has continued at intervals. This may be accounted for by some possible hemorrhagic invasion of the labyrinth.

Exacerbations Caused by Influenza.

Of seventeen mastoid operations performed by me in six consecutive days, during the second week of January, 1908, seven were of the recurrent variety. All showed extensive bone destruction, exposing either the sinus or dura or both. Two led to the evacuation of pus from a temporo-sphenoidal abscess, through the mastoid antrum route, and one assumed the Bezold's variety of mastoid disease. Previous duration of the disease ranged from twenty-seven months to thirty-one years. The acute exacerbations occurred in two cases about every two months, and in the others about every two years or more. Four of the patients had not noticed any discharge from the ear, nor experienced the slightest discomfort from the same for a period of three years or more. In one patient the ear had not discharged for six years when a sinus thrombosis developed. In all these patients the acute exacerbations were complications of influenza.

Of about one hundred mastoid operations recently performed, twenty-one were of the recurrent variety. Of this number, eleven showed unexpected necrotic exposure of the sinus or interior of the skull. Two died during the acute exacerbation, one from an old encysted temporo-sphenoidal abscess, which ruptured as the result of an injury to the head, and the other from an infectious meningitis. In this latter case free pus was found in the cavernous and petrosal sinuses, the lateral sinus being apparently uninvolved. This patient enjoyed good health until his final and fatal attack, his previous exacerbation occurring more than twelve years before. The primary ear disease developed from epidemic measles forty-two years prior to this date.

During an attack of suppurative otitis media, especially when long-continued, the mucosa lining the tympanic cavity and acces-

sory cavities first undergoes an ulcerative process, and this in turn becomes macerated and peels off, leaving a greater or less area of exposed osseous surface. With the protective and nutritive coat removed, the bone becomes an easy prey to the destructive influence of pathogenic micro-organisms. Consequently it is hardly to be wondered at that so much necrosis of the bony structure should occur in such a large number of suppurative cases.

The location of the perforation in the membrana tympani would seem to offer some points in the diagnosis of serious intratympanic as well as mastoid caries. Although the rule is not constant, it nevertheless does occur with sufficient frequency to warrant the assumption that perforations situated superiorly and posteriorly in Shrapnell's membrane, and more especially if they exist in connection with drooping of the posterior and superior wall of the external auditory canal, are definite diagnostic indications of extensive caries of the tympanic cavity as well as of the mastoid process.

During suppuration the mucous membrane of the tympanic cavity shows hypertrophy, with the formation of vegetation, or papillary growths, which may be large or small, discrete or confluent, extending along the inner wall of the cavity to the orifice of the Eustachian tube. As suppuration continues the epithelial cells desquamate and form large, whitish-yellow masses, which are composed of inspissated secretion and desquamated epithelium. These may be retained for an indefinite time unless infected by the staphylococcus, when they are converted into a foul-smelling discharge, consisting of bacteria and granular debris. Occasionally they assume a pearly white appearance and remain in the middle-ear cavity as homogeneous masses or cholesteatomata.

The inward growth of the epidermis of the external meatus towards the tympanic cavity without the formation of cholesteatomata takes place more often than is generally supposed. On the other hand, secondary cholesteatomata are very often caused by the intrusion of the epidermis of the external meatus into the middle ear. They also develop in the middle ear itself, as shown by the presence of non-nucleated squamous cells. These cholesteatomatous masses may attain a considerable size without the least sign of caries or necrosis. On the other hand, it must be remembered that these epithelial accumulations or growths are probably more de-

structive, both to the soft and osseous structures, and cause more serious mastoid and intracranial complications than any form of infection.

In long-standing cases the horizontal walls of the semi-circular canals may become eroded and the internal structures become the seat of inflammatory changes affecting the aqueductus vestibulae and the aqueductus cochleae. This in turn may cause a cerebellar abscess, resulting from suppuration within the labyrinth, the pus having followed the auditory nerve and burrowed its way through the internal auditory meatus.

More especially during epidemics of influenza we meet a large number of serious mastoid cases, the cause of which cannot be definitely explained. In other words, from the patient's history and in so far as can be seen, the middle ear was not involved. In cases of this kind, the bacteria and their toxins undoubtedly must have reached the mastoid by a hematogenous form of infection.

As is well known, recurrent cases may remain quiescent for some years, when the patient suddenly complains of severe pain in the head and ear of the same side. There is general hebetude, followed by chills, fever, vertigo and vomiting symptoms which frequently usher in some intracranial involvement.

In cases of a mild nature or where an intracranial complication is neither present nor in the course of development, the intensity of the above symptoms will be more or less modified, as the severity of the attack is governed entirely by the underlying involvement.

Diagnostic Value of Leukocytosis.

In considering the bacterial invasion of the temporal bone and the adjacent structures, we often speak of the presence and value of a leukocytosis. In this connection it is well to remember that a leukocytosis is a perfectly normal condition in the healthy state, and is, in fact, Nature's method of providing scavengers for the destruction of the numerous micro-organisms that are constantly invading the human economy. This leukocytosis, therefore, must necessarily vary in degree, depending entirely on the quantity and character of the toxins to be destroyed.

In the great majority of cases a normal leukocytosis is quite sufficient to destroy the hostile microbic incursion, but when the

pathogenic organisms are numerous and virulent, a battle royal ensues between the leukocytes, which resent the hostile entrance into the possessions or domains of another, and the invading microbic army. In order to successfully meet and destroy the enemy, Nature greatly increases the normal percentage of leukocytes, in which state the term hyperleukocytosis should be used, the antithesis being hypoleukocytosis or leukopenia, which naturally signifies an entirely different condition. The normal number of leukocytes varies from 6,000 to 10,000 to the cubic millimeter. Hyperleukocytosis indicates an increase to 15,000 or more, and hypoleukocytosis or leukopenia is a decrease from the normal amount.

An examination of the blood is often an important link in the chain of symptoms which goes to make up the diagnosis in a septic process, but this examination must consist of the differential leukocyte count as distinguished from merely determining the presence of a hyperleukocytosis on the one hand, or leukopenia on the other.

The condition in sepsis, in the majority of cases, shows a decided increase in the leukocytes, but this is by no means constant, for as great an authority as Grawitz states that "not rarely active inflammatory processes with suppuration may be present without hyperleukocytosis occurring."

Leukocytosis gauges the combative powers an individual possesses and can use against the infection by which he is attacked, but it does not show the strength of that infection. Thus, in trifling infections in which the activity of the leukocytes is insufficient, and also in fatal cases where the system is overwhelmed by the toxins, hyperleukocytosis does not develop and even leukopenia may be absent. Facts such as these render the occurrence of hyperleukocytosis in sepsis an inconstant phenomenon. An early development and rapid increase in the number of leukocytes is decidedly in favor of septic disease. We have, however, a good index to the severity of the septic process in the relative percentage of polynuclear leukocytes. Normally there is present a variation of from 59 to 68 percent of these cells, with a mean percentage of 61. Therefore, if a patient presents a blood-picture in which the relative polynuclear percentage is above 80, it indicates absorption of pus from some part of the body. Pus

is seldom found with a relative percentage of less than 80 unless the patient is a child, in which case it may be as low as 73. However, pus should be suspected and looked for in all cases in which the polynuclear percentage is above 70.

In addition to the hyperleukocytosis there is at times another element which may be determined from the examination of the blood, namely, the demonstration of the special micro-organism, often in almost pure culture. It is never sufficient to content ourselves with a single culture of the blood, but this should be repeated and in addition, various culture media should be employed.

The relations of the ear to sepsis are extremely important. Pontick examined the ears in one hundred children whose ages varied from one month to four years, and who had succumbed to various diseases. The autopsies revealed that 78 times there was bilateral, and 13 times unilateral, otitis media. In only nine cases was the middle ear normal.

From the foregoing it would seem that we have at our command a valuable asset, which, when properly worked out, may throw much invaluable light on certain forms of sepsis. For example, in cases of recurrent suppurative otitis media, during the stage of *apparent* inactivity; or in other words, between the periods that mark the acute exacerbations, it may be possible, by an examination of the blood, to demonstrate that a retrogressive metamorphosis is in actual progress. The value of such findings would at once become apparent if we were enabled thereby to appreciate such conditions, even in the absence of certain localizing symptoms

PAPER:

END-RESULTS IN 600 CASES OF OTITIS MEDIA PURULENTA CHRONICA.

J. E. SHEPPARD, M. D.

Of those that have discharged less than one year: Total..... 84
1. Cured in less than a month of treatment..... 51
2. Cured in more than a month of treatment 25
3. Cured by ossiculectomy 1
4. Cured by radical operation 2
5. Cured by Schwartze mastoid operation..... 1
6. Not cured by ossiculectomy, and as yet no further Op.... 1
7. Died after operation 3

Of those that have discharged more than 1 year, and less than 10 years 231
1. Cured in less than a month of treatment..... 62
2. Cured in more than a month of treatment 92
3. Cured by ossiculectomy 33
4. Cured by radical operation 26
5. Cured by Schwartze mastoid operation 1
6. Not cured by ossiculectomy, and as yet no further Op.... 7
7. Died after operation 2
8. Still under observation and discharging 8

Of those that have discharged more than 10 and less than 20 years 113
1. Cured in less than a month of treatment 28
2. Cured in more than a month of treatment 58
3. Cured by ossiculectomy 15
4. Cured by radical operation 5
5. Not cured by ossiculectomy, and as yet no further Op..... 2
6. Died after operation 1
7. Still under observation and discharging 4

Of those that have discharged more than 20 years.....142

1. Cured in less than a month of treatment 41
2. Cured in more than a month of treatment 66
3. Cured by ossiculectomy 13
4. Cured by radical operation 9
5. Not cured by ossiculectomy, and as yet no further Op.... 1
6. Died after operation 1
7. Still under observation and discharging..... 11

Granulations—	Per cent.
Present—	
Came to operation, 65	11.40
Did not come to operation, 168	29.47
Absent—	
Came to operation, 59	10.35
Did not come to operation, 278	48.78

Hearing improved 240
Hearing same 207
Hearing worse 38
Hearing, balance not known.
Vertigo present afterwards 58
Vertigo not present afterwards 292
Vertigo, balance no note.
Recurrences—did have 238
Recurrences—did not have 150
Recurrences—balance no note.
Male 277
Female 323

600

Persons suffering with both ears, 160 ..... Total 320
Persons suffering with A. D., 119 ..... Total 119
Persons suffering wth A. S., 161 ..... Total 161

600

Total deaths 7

Of 30 cases in which immediate operation was advised, but advice not accepted:
Suppurated, less than 20 years 11
Suppurated, more than 20 years 19

With granulations present, 15; absent, 15.

Recommendation for ossiculectomy	23
Recommendation for radical operation	7

SUMMARY.

	Per cent
423—Cured without operation	70.50
30—Operation advised first visit; advice not accepted	5.00
60—Cured by ossiculectomy	10.00
11—Ossiculectomy failed to cure, and as yet no other Op.	1.84
48—Cured by radical operation, Schwartze Op., etc.	8.00
7—Died	1.17
21—Still under observation, and discharging	3.49
600	100.00

Of the ossiculectomy cases:—

Facial paralysis	5
No facial paralysis	66
Radical operations Total	33
Cured so there have been no recurrences of suppuration	25
Recurrences (all of which have yielded readily to treatment)	8

So that I should call them all cured. This judgment depending on what cure means.

Facial paralysis present before operation	1
Facial paralysis present after operation	7
No facial paralysis	25

No other complications of moment.

Hearing—

Better	7
Same	7
Worse	7
No note	12
	33

What are the results of the operation?

1. On life?—Does not threaten it in my experience.
2. On the otorrhoea—Complete cure in 75.75 per cent of cases; cure with occasional recurrences in 24.25 per cent.

3. On the hearing—Better, 21.21 per cent; same, 21.21 per cent, worse, 21.21 per cent; no note, 36.37 per cent.
4. On the underlying bone disease—Cures it.
   15 operations—Mastoid and ossiculectomy.
5. Schwartze mastoid operations, for O. M. P. C. (1 case with facial paralysis before operation).
   8 cured without recurrences.
   1 cured with recurrences for awhile—final cure.
   Hearing better in 6 cases.
   Hearing same in 3 cases.
6. Schwartze mastoid operations with ossiculectomy—
   (1 case with facial paralysis before operation.)
   4 cured without recurrences.
   1 cured with recurrences for awhile, final cure.
   1 not cured, still under observation and discharging.
   Hearing better in 1, same in 4, worse in 1, no post-operative facial paralysis.

DEATHS.

1. Meningitis—diagnosed before operation—woman 64 years old—recurrent suppuration all her life. Final attack brought on by grippe.
2. Meningitis—diagnosed before operation—girl, aet. 7½ years. Recurrent pain and discharge 5 or 6 years. This attack began 1 month before I saw her.
3. Uncontrollable epistaxis 3 days after mastoid operation—Girl, aet. 9 years, mastoid operated by General Surgeon in November, and again in April, but never healed. I saw the case in September and operated on a very anaemic child—antrum had never been opened—whole mastoid tubercular—had already had one or two severe nose bleeds, and when the final one came, the most skillful treatment failed to stop it.
4. Male, aet. 15 years. Recurrent pain and discharge, 5 years. A. S. Developed cerebellar abscess, but repeated consultations made diagnosis of meningitis, and he was not operated. Autopsy showed cerebellar abscess, and only recent meningitis.
5. Male, aet. 7 years. Diagnosis of meningitis made when case was first seen. Had had suppurating ear 5 years without treatment. Operation failed to cure.

6. Male, aet. 32 years. Recurring pain and discharge 3 years. Was seen during acute recurrence, but all symptoms subsiding, except one, he was not operated in time, and developed meningitis. This one symptom was sagging of post. sup. canal, the importance of which was only then beginning to be discussed (January, '94), but I have never forgotten my lesson as to its importance.
7. Male, aet. 18 years. (Reported in Arch. of Otology, Vol. XXVI, No 3, 1897.) Lepto meningitis, and extra-dural abscess in region of foramen lacerum medius, involving also pituitary body.

Besides these, there were three other cases of intra-cranial involvement which recovered—two cases of serous meningitis, and one of cerebral abscess in posterior temporal lobes.

A peculiarity is the fact that in this series there was no case of lateral sinus involvement, which is so often said to be an accompaniment of chronic, rather than acute, middle ear suppuration. All of my cases of sinus involvement have accompanied acute middle ear suppuration.

DISCUSSION.

Dr. William H. Haskin, of New York City, expressed himself as emphatically opposed to the indiscriminate performance of either the Stacke or the Schwartze-Stacke radical operation. The radical operation was frequently performed for discharge, in the majority of which cases subsequent examination would show that the discharge was not cured. In many cases there was discharge due to tubal moisture, but he did not believe tubal moisture should be considered in deciding whether or not to perform the radical operation, as this could be cured by applications of nitrate of silver or other measures. The operation of Stacke, pure and simple, was really called for and should be performed only when definitely indicated, as outlined by Dr. McKernon. He agreed with Dr. McKernon with regard to hearing being only temporarily improved. Tinnitus was increased rather than decreased in the majority of cases. Otologists were not always honest in their reports concerning facial paralysis. It should not be said that these cases would recover after six weeks or two months, as a large proportion of them remained at least partially paralyzed. Reports with reference to fatalities were also absolutely false in many instances. The radical

operation was followed by death in a great many cases. So far as the cosmetic effect was concerned, large plastic operations upon the concha should be decried, the results in the majority of cases being no better than those attending a less radical major operation.

Dr. H. Holbrook Curtis, of New York City, asked Dr. Haskin if, by the Stacke operation, he meant the radical operation.

Dr. Haskin referred the question to Dr. McKernon to be answered in his closing remarks.

Dr. Stephen H. Lutz, of Brooklyn, said many cases returned to other clinics after being operated on in the various hospitals and discharged as cured, to be treated for discharging ears. He cited the case of a patient who had been operated on eleven times and "cured" twice, in New York and in England, who still had a discharging ear. He agreed with Dr. Haskin regarding hearing, tinnitus and facial paralysis. He also agreed that more deaths from radical operation occur than are reported. He deprecated the practice of telling patients that the operation is a simple one, which was certainly not true. He had had forty-four of these cases. Out of that number fourteen were complete cures; sixteen still had some discharge but refused further operation; four had recurrent discharge, but refused further operation, four were re-operated three or four times and cured; two died—not from operation—one by accident, the second died two or three years afterward from diphtheria, four cases disappeared before treatment was completed.

Dr. W. Sohier Bryant confined his remarks to the occurrence of complications in the various forms of suppurating ears. In a paper entitled, "The Management of Suppuration of the Middle Ear Based on an Analysis of a Series of 100 Consecutive Cases Seen in Private Practice" read before this society at the last annual meeting he showed that mastoid complications occurred in 20 per cent of the cases of chronic recurrent middle ear suppuration, in 25 per cent of perennial chronic suppurative cases, in 46 per cent of all forms of chronic suppurative cases combined, in 66 per cent of simple chronic suppurative cases, and in 71 per cent of the acute suppurative cases.

He had again gone over the record of this series of cases and found that the data gave the following approximate figures relative to the proportion of mastoiditis found in the various classes of suppurative ears. The length of time of the suppuration, the number of mastoid cases and their duration compared, gave the

following figures for the frequency of mastoid involvement in the various forms of ear suppuration, as these existed in the community, and also the proportionate probability that any one given case had or would have mastoid symptoms. These figures were arrived at by averaging the total number of days during which each case had had the ear affections. He found that in the perennial suppurative cases one in 2920 had mastoiditis; in all classes of chronic middle ear suppurative cases, one out of 1432 had symptoms of mastoiditis, and in acute suppuration, one out of every four cases had symptoms of mastoiditis; in the recurrent chronic cases one in 1217 cases had mastoiditis; and in the simple chronic cases, one in 164 cases had mastoiditis.

These figures showed in a graphic way that acute suppuration was 41 times more likely to be accompanied by mastoid symptoms than simple chronic suppuration was. Also that simple chronic suppuration was seven times more likely to develop mastoid symptoms than recurrent chronic suppurative cases were, and that chronic recurrent suppurative cases were one-sixteenth more likely to develop mastoid symptoms than all chronic suppurative cases combined are, and that all chronic suppurative cases combined were twice as likely to develop mastoid symptoms as perennial suppurative cases were, which have lasted over ten years, and that recurrent chronic suppurative cases were two and two-fifth times more likely to develop mastoid symptoms than perennial suppurative cases.

He thought these were two reasons why the recurrent type of chronic purulent otitis media was more likely to be complicated than the perennial type: First, because in reality the suppuration does not stop but becomes blocked in the cases that are not under expert observation; therefore the infection seeks other outlets than the meatus; and second, because by his classification many of these cases really belonged to the simple chronic type on account of their short chronicity. They had seen that the simple chronic type was very subject to complications.

In regard to intracranial complications, there were 11 per cent of his series affected; 8 per cent were of inflammatory nature. The inflammatory cases they distributed in this manner. No cases occurred in the recurrent variety of chronic suppuration. Eight per cent of the perennial, 9 per cent of the simple chronic cases and 10 per cent of the acute cases had inflammatory intracranial com-

plications. This was exactly the same order of frequency as for the frequency of mastoiditis. They would naturally expect to have the most intracranial complications where they had the most mastoiditis. The final conclusion was that the longer the suppuration lasted without mastoid symptoms, the less likelihood there was of the appearance of such symptoms. In the acute cases resistance was least and increased with the continuance of the prolongation of the inflammation.

Dr. George A. Leland, of Boston, agreed with Dr. Smith concerning the seriousness of chronic discharging ears and that this should be duly impressed upon the minds of the rest of the profession as well as of the laity. These cases should be treated by the aurist and not by the family physician. In many instances, mastoid involvement following colds or infections, external or internal, would be early recognized, i. e. before manifestations of the usual symptoms, or on the onset of perhaps fatal complications; or would be obviated by the maintenance of proper ventilation and drainage through the natural channels. Recurrent infection was often not diagnosticated because of the absence of a marked discharge from the ear, the subjective symptoms noted being slight fever, malaise, aprosexia, symptoms of malaria, etc. In the presence of large perforations, especially, there was always danger of infection from bathing in impure water, e. g., the dilute sewage of many of our coast resorts, and such ears should always be protected in bathing by a firm plug of non-absorbent cotton. With reference to complete exenteration, he took exception to some of the statements made, saying that he had yet to see a death caused by the operation. Some patients may have died after the operation,—one from nephritis, for example, which was not suspected at the time. He had seen no cases of meningitis. He had not seen tinnitus increased but on the contrary frequently diminished and the hearing had often much improved, not only where one but where both ears had been exenterated.

Professor Jansen, in discussing Dr. McKernon's paper, said: By the Stacke operation we understand, in Germany, the exposure of the tympanum by way of the attic. Usually, the radical exposure from behind is done, which, first published by Zaufall, was performed by me in Berlin entirely independently of Zaufall, and described by me, at the International Congress at Berlin, in 1800. We speak, therefore, of the chiseling open of the mastoid process

and antrum, after Schwartze, of the radical operation, and finally, of the Stacke operation. It does not seem to me to be proper to compare the time of healing, indiscriminately, in cases of radical operation. The cases are far too varied. We cannot compare, for instance, a case where the disease is limited to the antrum, with one where there is extensive involvement of the labyrinthine wall. The etiology is an important factor, whether due to scarlatina, or tuberculosis. Furthermore, it is of great importance in this question, to note the localization more accurately, in the diagnosis, as well as the etiology. The structure of the tympanum is also of great importance. Healing is retarded, where there is a deep recessus hypotympanicus or retro-facialis, where pockets easily form. Through careful exposure and smoothing down of these recesses, I have often achieved rapid healing, in cases of long continued suppuration, after a radical operation.

In cases of disease of the labyrinthine wall, there is often disease of the cochlea. Aside from this complication and tuberculosis, the Thiersch skin-transplantation causes a marked diminution in the time of healing, as well as making the treatment of the wound less unpleasant. The most favorable time for the performance of skin-graft seems to me to be immediately after the operation, or at the first dressing, several days later. I have also had good results, when it was done ten to fourteen days later. In some cases, in spite of the Thiersch graft, the cavity does not epidermatize; a slightly thickened membrane, with red moist surface, slightly granular and firm, covers the bone, and remains unchanged for months and years. The epidermis does not push its way over it. The underlying bone is diseased, usually becoming diseased during the after-treatment. This red membrane must be removed, as well as the diseased bone, down to healthy bone. With the help of a Thiersch graft, healing usually occurs rapidly.

Facial paralysis often disappears after six to nine months, and even after a year. It is usually due to an error of technic.

The causes of aggravation of symptoms and exitus, following an operation, may be the following: (1) A latent labyrinthine involvement may pass into the active stage. Through Barany's method we can now determine before the operation, whether the latent form is present. (2) An injury of the normal labyrinth, followed by infection, is not very uncommon. (3) An infection of the wound is rare. As these three causes may be thrown out, by

any one with the necessary experience, care and technic we may consider the operation as without danger.

In regard to functional results, I have had the following experience: Where there has been poor hearing, I have often seen considerable improvement. Where there was very good hearing, however, there is usually a diminution.

The ossicles are often entirely normal, as well as the corresponding portion of the tympanic cavity. Such cases, provided there is good hearing, should always be operated, with preservation of the membrana tympani and ossicles, in other respects, of course, according to the rules of the radical operation. We know that granulations often disappear after thorough exposure and drainage of the surrounding parts. This often occurs in the case of the large granulations which cover the ossicles. They gradually disappear, and in their place appears a thin, transparent membrane. The poor hearing is often improved considerably. In order to preserve good hearing, I have often done the radical operation, with preservation of the membrana tympani and ossicles. Whoever considers these structures to have an auditory function, will undoubtedly approve of my method. It is possible, in this way, to expose the upper part of the attic, and even (although this is difficult, and not always necessary) to smooth down the tegmen tympani. The lower, posterior portion of the tympanum can also be exposed, if necessary. I published this radical operation, with preservation of the membrana tympani and ossicles, in 1893 or 1894, at the meeting of the German Otological Society, and since then, have operated on 150 to 200 cases after this method, among a total of several thousand radical operations. The functional result was often excellent, although the after-treatment was often protracted. In some cases it was necessary to remove diseased ossicles later. It is absolutely essential for the joint between the long process of the incus and the stapes to be intact. About one and a half years ago Dr. Heath, of London, published this operation, as devised by him, at a time when, in Germany, a large number of otologists had a series of such operations to show. The too enthusiastic recommendation of this operation by Dr. Heath I cannot concur in. At all events, it is not Dr. Heath's operation, but mine. Occasionally there are accidents, such as luxation of the incus.

Dr. Edward B. Dench, of New York City, agreed with Dr. McKernon concerning the matter of preliminary treatment, and with Dr. Allport with reference to the care of the nose and throat. He had had no reason to change the views expressed in the paper to which Dr. Allport referred. The new integument lining the cavity must become accustomed to its habitat, but in the interim there would be a certain amount of secretion and a filling of the cavity with detritus which should be removed. In a year or two however, the cavity would be lined with skin which could take care of itself. He agreed with Dr. McKernon concerning the importance of the after-dressing. He had repeatedly emphasized this point at the New York Eye and Ear Infirmary and in private practice. In hospitals where the men had not had experience results were uniformly bad; where these cases were dressed by those having had experience results were uniformly good. This did not necessarily mean that cases in the first category came to re-operation, but it was necessary to use the curet and forceps. He emphasized the importance of careful asepsis for several days following operation in order to prevent the formation of granulations and the continuance of discharge. The formation of a moderately large meatus was imperative. In nine-tenths of the cases in which he had found re-operation necessary there was deficient drainage. It was not necessary to go entirely through the concha in order to make the meatus sufficiently large. In ninety-eight cases reported by him the mortality was two per cent. The hearing was improved in twenty-five per cent, not influenced in twenty-five per cent, and in only about two per cent was it made worse. He agreed with Dr. Jansen that where the hearing is exceedingly poor, if labyrinthine lesion can be excluded, the patient may safely be promised an improvement of hearing; but if the hearing is very good it will probably be made worse by radical operation. Dr. McKernon had stated that the hearing was made better immediately after operation but subsequently became worse; in his experience the hearing gradually improved as the stapes became more movable.

Dr. Wendell C. Phillips, of New York City, held that, regardless of its post-operative effects, the Schwartze-Stacke operation was absolutely necessary in certain cases. No surgeon would think of allowing a necrotic process in the hip joint or elsewhere to continue unchecked, and so it should be with the ear and mastoid process. In the presence of undoubted bone necrosis in this region

one must take the chances as to post-operative effects, which, to be sure, were not always what might be desired. An indication, not mentioned by the previous speakers, had always required operation in his experience, viz., atresia of the canal associated with the purulent process. A chronic necrotic process in the middle ear demanded operation because of the presence of necrotic bone which should be removed by surgical interference, not for the improvement of hearing. The patient should be made to understand that the improvement of hearing is not necessarily concerned. He did not agree with Dr. Leland with reference to mortality following this operation. Death was undoubtedly sometimes caused, or at least hastened, thereby.

The deformity resulting from a large opening could be obviated by properly made flaps.

Dr. Robert C. Myles, of New York City, said he had been searching the literature for whatever evidence he could find upon the subject under discussion and Dr. McKernon's paper was the first to give the positive and negative results of the operation in such clear-cut manner. A great deal was heard of the good results of the Schwartze-Stacke operation but the evil ones were as yet untabulated to a great extent. It was time correct statistics were forthcoming and Dr. McKernon had probably given the first definite report. The speaker had had but one death following operation of this kind and this he naturally attributed to the operation. There was some intracranial condition which could not be located, autopsy not being permitted. There was an abscess which was evidently metastatic and the center of the eye was filled with pus when opened.

Dr. Frank Allport, of Chicago, had no doubt that death occurred sometimes as the result of the radical operation, but believed that such occurrences were extremely infrequent. All operations were subject to fatalities but this did not deter the general surgeon from operating when surgical procedures seemed advisable. Neither should it deter aural surgeons from continuing the use of this great and admirable operation. These was no other way of logically handling cases of deep-seated necrosis of the temporal bone. The only thing for aural surgeons to do was to improve their technique and go on with the work.

Reference had been made to the continuance of a certain discharge which frequently followed the radical mastoid operation.

Dr. Allport had taken great comfort from the paper written by Dr. Dench some years ago on this subject and had come to the same conclusion arrived at by that distinguished surgeon. The skin which lines the mastoid cavity after the mastoid operation is thin and poorly nourished and frequently throws out a deposit on its surface which macerates and becomes unhealthy and purulent. Upon looking into a cavity of this kind the inexperienced surgeon feels that his work has been a failure, but such is not the case. In such cases the cavity should be thoroughly cleaned and soaked with bichloride solution, alcohol, or something of this nature, and this process should be repeated from time to time, keeping the parts clean and healthy until the skin is able to take care of itself.

It has been said that some surgeons make the radical operation too frequently. This may be so but Dr. Allport did not believe it to be with conscientious surgeons of experience. Cases of chronic purulent otorrhea should, of course, be subjected to proper and intelligent treatment for a reasonable time and their intractability practically assured before a radical operation is performed. Dr Allport believed that this was the rule adopted by conscientious experienced surgeons.

Dr. Allport believed that these cases did much better if nose and throat defects were relieved. He believed that many cases of persistent discharge were induced by adenoids or tonsils, for instance, which should be thoroughly removed before the radical operation is performed.

Concerning the hearing results after a radical mastoid operation, Dr. Allport believed with others that if the hearing was good before the operation it probably would be somewhat impaired by the operation, whereas if the hearing is poor before the radical mastoid operation, it will probably be improved by the operative procedure. He believed that the broad statement might be made that a large majority of patients have their hearing either improved or at least not injured by the radical mastoid operation and had, himself, quite a number of patients upon whom he had performed a radical mastoid operation on both sides, who had ears possessed with sufficient hearing to carry on an ordinary conversation as if nothing had been done to their ears.

Dr. Kaspar Pischel, of San Francisco, Cal., added to the causes of failure mentioned by Dr. Jansen's general anesthesia. Dr. A. D. Biven, of Chicago, had brought out the fact that three

or four days after operation death may occur from autointoxication. The principal feature was fatty degeneration of the liver. Chloroform was more dangerous than ether in this respect.

Dr. Norval H. Pierce, of Chicago, cited two cases of death resulting directly from operation. In one case, when he went into the antrum he uncovered but apparently did not injure the dura. The patient died from general septic meningitis. Autopsy showed that the labyrinth was intact. The route of invasion was absolutely unknown but the fact remained that the boy died of a general septic meningitis in five days. In the other case he accidentally uncovered the external semicircular canal to the extent of not more than a pin-head. The patient had some dizziness immediately following operation but improved greatly in the course of the next week and the bad symptoms did not become manifest until the packing, which had been carried out through the external wound, had been permanently removed. About ten days after operation, septic meningitis suddenly developed and the patient died. Post-mortem examination showed a septic inflammation of the meninges which had found its way through the internal auditory canal. Whether in the first case, death was due directly to the operation or was merely co-incidental he could not say, but in the second case it was undoubtedly traceable directly to the operation. With reference to the influence of the radical operation upon hearing, all were more or less remiss in the matter of examining these cases previous to operation, with regard to the auditory function, the motility of the stapes, the condition of the labyrinth, etc. Operation did not materially make this hearing worse, after two years, in many cases. Of the cases which had been carefully examined before operation, he had been able to control only half and in these the majority heard as well, and in some instances better, than before operation. A large number, however, heard distinctly worse.

Dr. J. A. Stucky, of Lexington, Ky., believed no subject in otology to be more important than that under discussion. The pendulum was swinging from one extreme to another, but the happy medium he believed had been found. So far as chronic suppurative disease of the middle ear was concerned he felt sure that all were agreed that radical conservatism is as dangerous as conservative radicalism is safe, and that it is better to operate a day too soon than an hour too late. He was loath to believe that any otologist would do a radical operation upon a patient so long as

there was reasonable ground for thinking a cure could be effected without operation. Operative treatment was frequently unsatisfactory because of failure to give the thorough drainage that is always necessary. He was glad to hear Dr. Smith emphasize the importance of co-operation on the part of the general surgeon and the otologist. He was surprised at the percentage of failures and partial successes reported by Dr. Smith and Dr. McKernon. In more than a hundred cases where tinnitus existed, it was usually relieved or markedly benefited by operation. He had had no deaths attributable to the operation, nor had he had any cases of permanent facial paralysis due to the operation. The hearing was made no worse in any case, but in five cases it was markedly improved. He had no explanation to offer on this point. Dr. Pierce's point concerning the examination of the ear was important.

In closing the discussion Dr. Stucky exhibited a pair of small forceps for removing the remnant of ossicles in the radical operation, also a little hand drill which he had found helpful in getting rid of the moisture in the Eustachian tube.

Dr. Joseph Beck, of Chicago, had made fairly careful tests with reference to the hearing before and after the radical operation and he had found uniformly that the hearing was lessened. By this he meant the ability to understand conversation. These patients could always hear the watch test but could not understand when spoken to; there being a distinct difference between *hearing* and *understanding*. He had also investigated the matter of facial paralysis following radical operation and had found that many cases of facial paralysis recorded were not paralysis due to injury of the facial nerve *per se*, but a neuritis, the time of appearance of which was important. Paralysis appearing slowly was not of so much importance as that which comes immediately after recovery from the anesthetic. In this experience, the majority of cases of chronic suppuration operated upon were clinical cases, which it was impossible to treat conservatively with any degree of satisfaction and consequently early operation was resorted to.

Dr. McKernon, in closing the discussion, said in reply to Dr Haskin's remarks that in a large proportion of adult cases the Stacke operation was sufficient and not the Schwartze-Stacke. In young adults and children it was extremely important that the mastoid process be explored at the time the radical operation was done. He did not agree with Dr. Haskin that it is rarely necessary

to do the Schwartze-Stacke operation. He did not, however, wish to be understood as opposing the Stacke operation whenever the indications therefor were definite. In the event of dead bone being present in or adjacent to the tympanic cavity, the latter operation should be resorted to. Dr. Lutz advocated the removal of diseased adenoids at the time of radical operation; in this the speaker did not concur. Dr. Leland and Dr. Bryant had found tinnitus to be lessened; this had not been his experience. In a certain proportion of cases it had been lessened, but where there was a formation of scar tissue in and around the oval window, it had been increased. He agreed with Dr. Phillips concerning the deformity resulting from a large meatal incision. Dr. Allport spoke of the radical mastoid operation, whereas the paper dealt with the Stacke operation. Referring to Dr. Jansen's remarks about the tubercular manifestations, he said five of the cases reported had tuberculosis of the middle ear at the time of operation, whether as the result of injury during, or of infection following operation, he was unable to say. Replying to Dr. Stucky's remarks concerning audition, all cases had been given a functional test following operation and at intervals for two years thereafter, so that the speaker could give a definite statement regarding the cases which had come under his personal observation. He had never noted the influence of autointoxication, as suggested by Dr. Pischel.

Having been asked to give a brief statement, in closing the discussion, of the routine after-treatment following the radical operation, Dr. McKernon outlined it as follows: After complete removal of every vestige of the disease the cavity is packed either with plain or iodoform gauze, the latter being used when the dural area is exposed, in other cases the former.

If there is a little or no moisture from the canal this packing is removed at the end of the third day, otherwise at an earlier time. After removal of the dressing the surface is powdered over and covered with plain sterile gauze. After the third dressing no more gauze is packed in. The stitches are removed at the end of the fourth day.

Dr. S. J. Kopetzky, of New York City, emphasized the importance, in the study of recurrent mastoiditis, especially after operative procedure, of differentiating the underlying condition. He knew of no satisfactory statistics which give the results of the radical operation based upon the underlying condition. The majority of published

statistics only seemed to take the cessation of the otorrhea into consideration. In other words, the radical operation was judged by the effect it had on a symptom only, and the operation was a failure if the otorrhea persisted after operation and was considered a success when the otorrhea was suppressed, irrespective of whether the disease causing the otorrhea had or had not been eradicated. His experience showed that in the majority of instances, wherein the suppuration persisted, the otoscopic picture, before operation, had shown a perforation of varying size which did not encroach upon the annulus tympanicus, in other words, a non-marginal perforation. This finding of a non-marginal perforation he interpreted, therefore, as evidence of a chronic disease of the lining mucous membrane of the middle ear, taken together with other significant data, cyto-diagnosis of the ear discharge, bacterial examination, etc. The radical operation, no matter how thoroughly carried out, did not remove all the diseased membrane, sections of which naturally remained in situ at such points as the oval window and the unapproachable depths of the Eustachian tube orifice. Therefore while the radical operation laid the middle ear open to subsequent after-treatment, in case of disease limited to the lining mucous membrane, it naturally failed to stop the otorrhea. Such cases should therefore be differentiated and not counted as increasing the percentages of failure from operation. On the other hand, he did not wish to be understood as holding that every non-marginal perforation excluded the presence of bone necrosis. There were mixed cases. In cases where bone necrosis was diagnosed, the results from the radical had proved good in proportion as the necrosed tissue was radically removed. The actual disease of the bone which resulted in the necrosis was too often entirely overlooked. The underlying tuberculosis, syphilitic or other disease was insufficiently understood by the medical profession at large. The summing up of Professor Jansen, together with what Dr. McKernon had so well presented in his paper, made it clear that the radical operation has distinct value in bone necrosis, and gives excellent results when thus indicated.

Dr. Smith, in closing the discussion, said that the majority of these cases would have a recurrence every few weeks, while others would not suffer from an acute exacerbation for some years. Patients of the latter class had heretofore been looked upon as of little or no importance, yet these cases, in his opinion, which was borne

out by an extensive experience, showed more carious destruction of bone and more frequent exposure of the underlying structures, more especially the sinus and dura, than is found in a given number of patients suffering from a continuous form of discharge. The dangers attendant upon a discharging ear cannot be too strongly emphasized. It is our duty, therefore, to present this matter in a way that will impress its importance upon the medical profession.

Dr. Smith was surprised to find that the death rate had been so high, in the experience of some of the Fellows, following the radical mastoid operation, and felt that this must be the result of patients being virtually in a moribund condition, due to some intracranial complication, before being subjected to operative interference, as he considered that conservatism was best served by advising an operation in the great majority of all chronic suppurative diseases of the ear, either of the recurrent or continuous variety, when the adoption of more simple means had failed to bring about a cure.

In regard to the continuation of a mucous secretion from the ear, following a mastoid operation he feels that this is practically always due to failure to close the tympanic orifice of the Eustachian tube at the time of operation. However, even though this should persist indefinitely, it is a matter of little importance, except from an esthetic point of view, from the fact that the operation has relieved the subject from all danger so far as life is concerned. Furthermore, it is a matter of little importance to subsequently close the tympanic orifice of the Eustachian tube, under cocaine anesthesia, through the external auditory canal. This he believed was best accomplished by the use of Allport's burrs.

In cases with good bone conduction, where the hearing was fairly good before operation and had been impaired as a result of operative interference, he believed this loss of hearing to be due to an exudate thrown out about the oval and round windows and the formation of adhesions resulting from the same, thus preventing the functional activity of these important parts, whereas in a similar case in which the hearing had materially improved after the operation, this probably could be accounted for from the fact that certain adhesions, the result of a former exudate, had been removed at the time of operation, which had the effect of liberating the stapes and removing the obstruction covering the round window, thereby restoring their normal function.

SYMPOSIUM ON THE CLIMATE OF THE UNITED STATES IN ITS ETIOLOGIC AND THERAPEUTIC RELATION TO DISEASES OF THE NOSE, THROAT AND EAR.

PAPER:

THE CLIMATE OF THE NORTH AND NORTHWEST IN ITS ETIOLOGIC AND THERAPEUTIC RELATION TO DISEASES OF THE NOSE, THROAT AND EAR.

CHARLES NELSON SPRATT, B. S., M. D.

"The climate of a region or site is the combined effect of the atmosphere and of the nature of the surface, in their relation to man." The weather is merely a phase of the climate, changeable from day to day.

The atmosphere varies in chemical composition, in temperature, in pressure, humidity, diathermancy, transparency, dust electrical conditions, and in the force and direction of the winds. Other factors are latitude, altitude, relations to large bodies of water, deserts, etc., nature of the soil, surface, i. e. slopes, plains valleys, etc., cultivation of the soil, drainage, manufactories and density of population.

The untutored savage has always looked upon the natural phenomena with awe and reverence. It is not strange that to-day, certain individuals hold the climate responsible for their aches and pains. They likewise think of some climate, other than their own, generally those described by railroad circulars, as the panacea for all their troubles.

As knowledge replaces theories, the direct influence of climate in the etiology and treatment of disease, becomes less prominent.

Twenty years ago malaria was considered to be a direct result of climate. A certain "miasma" arising from swamps was thought to be the cause of the disease. We now know that malaria is

due to a protozoon transmitted to man by the mosquito. Until recent years yellow fever and the "sleeping sickness" were considered to be in some way caused by the tropical climate. Reed, Lazear and Carroll demonstrated beyond doubt, that the infection of yellow fever is likewise transmitted to man by the mosquito. Koch has recently shown that the sleeping sickness of Africa belongs in the same group. The hot climate offers conditions favorable for the existence and spread of these diseases. Typhoid fever, influenza, pneumonia and many other diseases formerly considered to be due to climate, are now known to be caused by bacteria.

Although man can exist in any climate, hot or cold, moist or dry, high or low, where food is sufficient to support life, or the natural enemies are not too numerous, his physical, mental and moral make-up are influenced by the external conditions.

Experimental work on animals has shown that chilling, either by ether spray, or dipping into ice water, is generally followed by cloudy swelling, and granular degeneration of the hepatic and renal cells, with fever and albuminaria. While these experiments are not applicable to man, they show that excessive changes in temperature will cause, at least in animals, pathologic conditions. Lode found that animals chilled by wetting the fur more readily succumbed to infections. This may explain why pneumonia, rhinitis, and other acute inflammations of the respiratory tract, that so frequently follow a chilling of the body, are due to infection following a lowering of the resistance.

The climate of the north and northwestern part of the United States, east of the Rocky Mountains, would be classed, according to Koppen, as a cool temperate climate, i. e. monthly averages above 68° F. are limited to the summer months.

In such a vast territory, extending nearly 2000 miles from east to west, and 500 miles from north to south, with the same general climate there are many variations. In a paper limited as to time it will be impossible to speak more than in general terms of these minor differences in climate. The climate of a certain locality is often quite different from regions only a few miles distant. A southern exposure is generally warmer and more protected from the winds than a northern, valleys are generally damp, the air of the hills and mountains is dry.

The winters of Minnesota and Northern Maine are long and rigorous, the summers short and often sultry. The winters in

regions 400 miles south are mild, the summers long and semi-tropical.

The mountain ranges along the Atlantic coast offer a cooler and drier climate than regions near the ocean.

The amount of sunshine, in different portions of the North, varies considerably. In the winter of 1901 at Buffalo, New York, there were 56 cloudy, 34 partly cloudy and no clear days, while at Atlantic City there were 22 cloudy, 32 partly cloudy and 26 clear days. In general, along the Atlantic coast, the sunshine increases as we go south and the fog becomes less.

The prevailing winds are from the Northwest and South. The former are dry, the moisture having been precipitated on the western slope of the Cascade Mountains. The moist south winds, a part of the trade winds, travel from the Gulf up the Mississippi Valley. The New England coast in the winter time is often swept by cold damp gales from the North and Northeast. Fogs so frequent along the Atlantic coast are rare in the Northwest

The so-called dry steady winters of the Northwest show as great a daily range of temperature as in the East, but it is less noticeable to the individual. A drop of 20° in temperature, from 50° to 30° F., is more noticeable than an equal change of temperature from 20° to zero.

The humidity varies considerably, being greater near large bodies of water. The climate of the Northwest has for a long time enjoyed the reputation of being dry. This, according to weather reports, is more apparent than real. Owing to the lower temperature, the absolute humidity of the Northwest is less than the Eastern States. The mean relative humidity is about the same as that of the middle Atlantic coast. Below 55° F. the evaporation of cutaneous moisture is so slight that the humidity is not noticeable.

With the exception of the mountains along the eastern coast, the elevation is for the most part under 1000 feet.

It is undoubtedly true that a large percentage of the inhabitants of the northern part of the United States are sufferers from what are commonly grouped under the indefinite but comprehensive term "catarrh." It is well known that acute inflammation of the respiratory tract, the "colds," the "coughs" and the "sore throats" are not as frequent in the summer months as during the

rest of the year. Nasal stenosis and excessive secretion are more common in cold weather.

This is due to the extra work thrown on the nose in warming and moistening the cold, dry air. Inturgescence of the turbinates occurs if the nose is narrow or deviations or spurs exist; stenosis results and this is followed by an increase in the amount of nasal discharge.

It is also true that individuals with adenoids, hypertrophied turbinates, chronic pharyngitis and laryngitis, are worse during the winter months. That these diseases of the respiratory tract are not due to cold *per se* seems probable from the fact, that the original inhabitants and early settlers were relatively immune.

A cold climate is bracing and adapted for the maintenance of vigorous health. The activity of the individual is greater, more CO_2 is said to be given off by the lungs and muscular power is increased. With moderate changes in temperature the individual's resistance is increased.

In considering the effects produced by climate, in its relation to disease, we must take into consideration a far more important factor,—the artificial climate. In the northern part of the United States, the individual lives for nearly eight months under the unnatural conditions of this artificial climate. In the East and Central States, five or six months are spent in the natural climate, the remainder in the artificial.

This artificial climate is an outgrowth of our modern civilization. By this I do not refer to the changes produced by the drainage of swamps, the felling of forests, etc., but to certain far more important changes; of these may be mentioned:

First, Crowding.—According to the United States census of 1900 31.3 per cent. of the total population lived in cities of 8,000 or more. This herding together increases the spread of infection, especially diseases of the respiratory tract. As a rule the denser the population the more unsanitary are the conditions.

Second, Dust.—In the city the pure air of the forest and prairie is replaced by a smoky, filthy, dust laden atmosphere. Dried sputum and excrement from many horses, pulverized by the constant traffic, thrown into the air by street cars and automobiles, is blown into our eyes and nostrils. No window casing is tight enough to keep it out of our buildings. The constant breathing of such dust laden air, even if the bacteria have been

killed by the action of the sunlight, must have an irritating effect on the respiratory mucous membrane.

Pneumonia, influenza, and tuberculosis are in many cases dust infections. Could the infection be traced, doubtless acute rhinitis, tonsilitis, conjunctivitis, might be included in the above. Polishers stone-cutters and grinders, are frequently sacrificed to their occupations, by the constant inhalation of sterile dust.

Health officers say that after a few bright, warm, dry, dusty days, the number of cases of infectious diseases is sure to be increased. The severe influenza epidemic of 1889 followed a mild open winter and autumn. The same is true of the past winter in the Northwest. There is much truth in "A green Christmas means a fat graveyard."

Third, Overheated buildings.—As has been stated, during six to eight months of the entire year, a large percentage of the population live in a semi-tropical climate. Houses and offices are heated by steam, hot air, etc., until the temperature is 70° F. or over. The humidity in most cases ranges from 5 per cent. to 10 per cent., when it should be 50 per cent. to 70 per cent. In zero weather to give a relative humidity of 65 per cent. would require in the average house with a temperature of 70° F. the evaporation of six gallons of water.

Fourth, Sudden and marked changes in temperature and humidity.—In the natural climate a fall of 20° in temperature as from 70° to 50° F. is very noticeable, an equal fall from 20° F. to zero is not so apparent. Although in the Northwest the changes in temperature are just as marked as along the Atlantic coast, the changes in the winter are not so noticeable on account of the low average thermometer. In the winter months the changes from the hot, dry air of our buildings to that of out-of-doors may range from 30° to 100° F. The suddenness of this change is not to be compared to that of any natural climate. The effect of this change on the respiratory tract, is far more serious and likely to cause irritation than cold.

Fifth, Deficiency of Sunlight.—This is due not only to the thick pall of smoke covering most of our cities, but to the indoor life led by a large proportion of our population. The bactericidal action of light is an important factor in preventing the spread of disease.

Pneumonia, influenza, acute sinusitis, diphtheria, acute otitis media, tonsilitis and probably acute laryngitis, rhinitis and pharygitis, are due to bacterial infection. Sudden changes in temperature, dust, and mouth breathing, lessen the resistance and make infection possible.

Repeated attacks of acute inflammation, especially when the individuals live under unnatural conditions, lead to permanent changes in the tissues. We thus have hypertrophy of the turbinates, adenoids, enlarged tonsils, chronic sinusitis, polypi, and chronic inflammation of the larynx and pharynx. A large percentage of chronic catarrhal otitis media cases are secondary to diseased conditions of the nose and naso-pharynx. The tinnitus accompanying such conditions is in some cases undoubtedly influenced by the atmospheric changes. These cases frequently improve in hearing and the tinnitus ceases after free nasal respiration has been established by operation. Practically all cases of hypertrophy of the turbinates, adenoids and enlarged tonsils, cases with dry and the secretory form of catarrh of the middle ear are better during the summer months. The cold or rather the sudden changes from hot dry houses to the cold winter and autumn air aggravate the conditions. Patients who go south for the winter find relief.

True hay fever or rose cold, occurring in May and June, is not common in the United States. Our so-called hay fever occurs in the late summer and autumn. There can be but little doubt that in a large percentage of cases the pollen-toxin of the rag-weed or Ambrosia is the exciting factor. Britton and Brown give the distribution of the genus Ambrosia as follows: From Nova Scotia to Florida west to British Columbia.

In the North the plants are poorly developed and the amount of toxin is less than in those growing farther south. The plants bloom about the twentieth of August in the region about New York City. Sufferers find relief in the mountains and the north shores of the Great Lakes. The disease is rare in the region of Lake Superior.

Tuberculosis of the larynx is a disease practically always secondary to pulmonary tuberculosis. Cures of the latter condition are obtained in almost any climate. A moderately cool, dry climate with much sunshine has a more stimulating effect than a warm climate. Climate plays no role in the etiology of tuberculosis of the larynx. In regard to the treatment, the general resistance

must be increased and a mild climate with a dust free air is to be desired.

Acute diseases of the upper air passages are not subject to climatotherapy except as to prophylaxis. The chronic diseases of the upper respiratory tract, as adenoids, hypertrophic rhinitis, inturgescence, etc., cause less disturbance in a mild warm climate, not subject to sudden change.

The climate of the North, while both directly and indirectly being of much importance in the etiology of disease, has but little value in the treatment.

Conclusions—

First. The influence of climate in the etiology and treatment of disease has been over-estimated.

Second. Acute and chronic inflammation of the respiratory tracts are more frequent and worse during the winter months.

Third. These conditions are not due to cold but to certain artificial conditions under which the individual lives from six to eight months,—the artificial climate. This includes crowding, dust, over heated rooms with dry air, sudden changes from heated rooms to the cold outside air and deficiency of sunlight.

Fourth. Diseases of the ear, nose and throat are common in the North, due to the above conditions.

PAPER:

THE CLIMATE OF COLORADO ETIOLOGICALLY AND THERAPEUTICALLY CONSIDERED.

JAMES ALLEN PATTERSON, M. D.

The consideration of the various influences of the climate of Colorado upon the catarrhal condition of the upper air tract cannot be considered rationally without first briefly alluding to its varied physical geography. A state with an area of 103,925 square miles with watersheds running in diverse directions, altitudes varying from 4,000 feet to forty mountain peaks over 14,000 feet and fifty over 13,000 feet, naturally presents different characteristics.

What we term the western slope from the great continental divide in the neighborhood of Leadville at 10,500 feet to the Utah state line where the elevation lessens to 4,500 feet, presents a climate characterized by much snow in winter and in summer, heat distinctly above temperate. In this belt, in the valleys, sugar beet growing and fruit raising are carried on to great profit. On the eastern side of this slope when the higher mountain ranges are left behind, the climate is characterized by winters less severe with less snow and less rainfall and by summer nights too cool to foster the agricultural pursuits of the region just mentioned. Then as we go farther east or north and get away from the mountain influences, greater warmth in summer is encountered fostering, where water is available for irrigation purposes, productive agricultural pursuits.

The climate which is usually had in view when that of this state is mentioned in connection with the cure of tuberculosis, is that of this eastern slope in the foot-hills of the Rocky Mountains not far enough away to get the heat in summer, the cold in winter nor the atmospheric moisture of the states which border it on the east

In tubercular affections, I am impressed first by the importance of the climatic influence upon the laryngeal manifestation. I shall

never forget the fear in attempting the control and betterment of laryngeal tuberculosis engendered by experiences on the eastern seaboard that assailed me in beginning work in this line in Colorado, but I will remember the surprise that awaited me as a result of my efforts. I began seeing cases that I should have considered utterly hopeless, improving under methods that were futile elsewhere. I recall a case that presented the typical pyriform-shaped swelling of the arytenoids, hoarseness and much cough, go out of my observation for four of five weeks to sojourn upon a ranch in mid-winter where the food was supposed to be poor as well as plain, where one could live in only comparative comfort, where fortunately there was nothing to do but sit along the "sunny side of the barn," eat when food was served and sleep when the sun set. This person returned at the end of the period mentioned with arytenoid cartilages of normal contour, only some catarrhal laryngitis remaining. He is practicing his profession to-day—some eight years since. Yet I do not want you to deduce that our climate has an effect upon the larynx only; there may be a few exceptions to the rule, that laryngeal tuberculosis improves in more or less ratio to the general improvement of the patient's condition. When the patient is able to support the tubercular invasion which is aided by climate and hygiene, our well accepted methods of dealing with tuberculosis of the larynx are surely much enhanced.

Tubercular diseases of the middle ear as evidenced by breaking down of the drum membrane with little inflammatory or sensory symptoms are more apt to become quiescent, less liable to dangerous outbursts than elsewhere; and upon the improvement of the patient's general health or stay of his disease, the ears usually become free from discharge. Tuberculosis of the pharynx and mouth are so surely manfestations of the last stage of the disease that they are mentioned only to indicate the bad prognosis.

The influence of the dry air of Colorado upon catarrhal condition of the upper air passages is more difficult to summarize than its influence upon tubercular conditions.

Hypertrophic nasal catarrhs with more or less nasal stenosis are distinctly benefited. Where there is abundant secretion it is usually lessened so quickly on one's advent that a preliminary dryness as pointed out by the late Dr. Solly, with probably a little blood stain when the nose is blown, is frequently met with and leads

the patient to very erroneous conclusions regarding his affection, in fact a great deal of the mild nose-bleed which the laity attribute to high altitudes is simply due to inspissated mucus within the nasal vestibule that is picked off roughly until the mucous membrane is abraded and bleeds under a very slight irritation such as the blowing of the nose.

Those who suffer from true hay fever in other and damper climates are frequently absolutely relieved. I can attest personally to my own perfect cure of it since residing in Colorado Springs. Paradoxically there are found old residents who have developed hay fever in Colorado who do not have it in damp climates.

Atrophic rhinitis and its sometime accompaniment atrophic pharyngitis being dependent upon diverse causes is susceptible of so many classifications that I approach the subject with diffidence. I shall exclude ozaena. I commonly meet it as a very mild form of rhinitis sicca with sensations of annoyance far out of proportion to the variation from normal appearances. I have never seen this form progress unfavorably, though it is very rebellious to treatment except where it has been caused by irregularities of the septum, turbinal occlusions, or enlarged tonsils; in the latter cases great relief is obtained by operative procedures. These rebellious cases are usually found in lithaemic individuals and seem to be aggravated by the rapid evaporation of moisture in the anterior third of the nostril, which takes place in dry atmosphere. Such cases are distinctly more comfortable in moist climates.

Perforations of the septal cartilage, particularly where there is a deflection with occlusion, are more apt to occur than in damp regions; also septal deviations, encroaching upon the vestibule, are usually more apt to give annoyance than they do in moist zones.

Catarrhs of the Eustachian orifice, when due to hypertrophic rhinitis, are benefited; but since most of such cases are complicated with deflected septae, enlarged tonsils or even adenoids, operative measures are imperative.

I have been unable to formulate any rule to apply to the cases of chronic middle ear catarrh; I meet cases where the presence of tenacious mucus in the tube is most obstinate even where there is little abnormality of shape or where there is very little alteration in the secretion as evidenced by the appearance of the nose and nose pharynx. Many of these cases are benefited when coming from moist climes. My observation of the effect of treatment upon such

conditions originating in Colorado shows about the same degree of stubbornness everywhere encountered. I feel, however, that such cases developing in moist climes are benefited. Some of those developing in Colorado may be improved elsewhere, yet most of the cases that I have sent away have returned showing no benefit.

Cases are seen of chronic suppurative otitis media that come to Colorado, of their own accord, yearly, because they find the discharge lessens and their general condition is greatly improved by doing so.

My experience has been when operative measures are undertaken upon such cases that post-operative healing is unusually rapid. The deduction is therefore obvious. This is simply in accord with a statement made by Solly in 1903 that "with regard to surgical cases treated at an altitude, nasal or otherwise, it is undoubtedly true that they do better and require less post-operative care than at a low level."

DISCUSSION.

Dr. H. Bert Ellis, of Los Angeles, Cal., thought it mattered little in what portion of the country one live so far as catarrh was concerned. Other factors than changes in temperature entered into the condition. The more primitive the method of living, the less catarrh found.

Dr. Thomas J. Gallaher, of Denver, Colo., referring to the word "catarrh," called attention to Bosworth's criticism of the term. He also spoke of Bosworth's classic work in which he pointed out the difference between so-called "catching cold" and absolute cold, and emphasized the importance of bringing about an immunity to this and other acute conditions by proper methods of living. Relief from hay fever seemed to depend entirely upon the patient's getting away from the pollen to which he is susceptible, no matter in what climate that may be. Persons going to the dry climate and high altitude of Denver experienced some inconvenience at first because of turgescence of the turbinated bodies, but the equilibrium was soon established. Most pathological conditions of the nasal cavities were amenable to surgical interference.

PAPER:

THE VALUE OF THE DIFFERENTIAL BLOOD COUNT AND OF THE BACTERIOLOGICAL EXAMINATION OF THE AURAL DISCHARGE AS DIAGNOSTIC FACTORS IN CASES OF MASTOID INVOLVEMENT.

EDWARD BRADFORD DENCH, M. D.

The question as to the value of the differential blood count, and of the bacteriological examination of the aural discharge, as affording a definite factor in the prognosis of cases of suspected mastoiditis, has interested the otologist for a considerable period. From a general surgical standpoint, we would naturally expect that the presence of pus in the mastoid would cause not only an increase in the number of white cells circulating in the blood, but also an increase in the percentage of polymorphonuclear cells. This view was taken by most observers for a number of years after the value of the differential blood count became recognized in general surgical practice.

In 1907, the writer presented a paper before the New York Clinical Society, on the value of the differential blood count and of the bacteriological examination of the aural discharge, as an indication for the mastoid operation. This paper was published in the *New York Medical Journal*, October 12th, 1907.

At the suggestion of our Secretary, I have taken the liberty of bringing this subject to your consideration. The present paper will include the paper already published, together with added observations which have been made during the last twelve months.

Let us consider first the value of the differential blood count in cases of suspected mastoiditis. Referring to my former paper, I reported twenty-four cases, operated on at the New York Eye and Ear Infirmary. In seventeen of these the simple mastoid was performed, in six the radical operation was performed, owing

to the fact that acute symptoms occurred in a patient already suffering from a chronic middle ear suppuration. In one case, the mastoid suppuration had involved the cranial cavity, and an epidural abscess was found at the time of operation. The blood count in these twenty-four cases showed that in uncomplicated cases, the highest leucocytosis was 25,200, with a polymorphonuclear count of 64 per cent. In the other cases, the leucocytosis ranged between 6,000 and 20,000, and in no case did the polymorphonuclear count reach 80 per cent, in the uncomplicated cases.

The later statistics show that in thirty-six cases, in no uncomplicated case, did the polymorphonuclear count rise above 80 per cent. In seven cases, it was below 60 per cent, in eight cases between 60 per cent and 70 per cent, and in thirteen cases between 70 per cent and 80 per cent. In eight cases, following the mastoid operation, in which either some visceral, intracranial complication or infection of the superficial tissues took place, the polymorphonuclear count was above 80 per cent.

In all of these cases, of the last series, however, in which the polymorphonuclear count was above 80 per cent, the mastoid operation had already been performed, and the polymorphonuclear count was simply indicative of some further complicating lesion.

It is interesting to note, in this connection, that two cases, in which the polymorphonuclear count was above 80 per cent. recovered perfectly after incision of the drum membrane, while in two other cases—one in which the jugular was excised for thrombosis, and in another in which there was a complicating pneumonia, which later required excision of the jugular—the polymorphonuclear count never reached 80 per cent.

Taking up next, the leucocyte count, in eight cases the leucocyte count was below 9,000, in thirteen cases between 9,000 and 12,000 in ten cases between 12,000 and 20,000, and it was above 20,000 in four cases. One of these four cases died of meningitis; the second was a case of simple mastoiditis, in which after operation the polymorphonuclear percentage suddenly rose to 88 per cent with a white count of 40,000. The next day, however, the polymorphonuclear count was 74 per cent and the white count 16,100. Both the polymorphonuclear rise and the white blood count were undoubtedly due to some sudden visceral complication. In the third case, the increase in the polymorphonuclear count must be attributed to a similar cause, as two days later the polymorphonu-

clear count fell to 61.2 per cent, and the white cells to 13,000. In the fourth case, the white blood count was 29,800 in a case of simple mastoiditis; the polymorphonuclear count was only 71.4 per cent in this case.

I have simply given these four cases to show how misleading a single blood count might be.

Referring to the preceding paper, already quoted, it was found that in 24 cases the leucocytosis ranged between 6,000 and 20,000, in uncomplicated cases. In two cases, however, in one of which there was a burrowing abscess of the neck and later a cerebellar abscess, there was a high leucocytosis, of 33,000, with a polymorphonuclear count of 84 per cent. In a second case, one of double mastoiditis, complicated with suppuration of the supraclavicular glands and involvement of the lateral sinus, the leucocyte count at one time was 30,000, while the polymorphonuclear count at times ranged slightly over 80 per cent.

We may say, therefore, that in no single case of this series, did the differential blood count offer the slightest help in diagnosis. In the paper previously mentioned, I called particular attention to a case of acute otitis media, involving both ears, in which the polymorphonuclear percentage was about 84 per cent, while the leucocytosis was 29,000. In this case—that of a rather nervous boy—considerable mastoid tenderness was present. Both ears were draining freely, however, as the result of myringotomy, and the prompt appearance of certain signs in the chest showed that the increased leucocytosis and high polymorphonuclear percentage were due to the thoracic lesion, rather than to mastoid involvement. This patient made a perfect recovery, without any further operation than myringotomy.

When we come to consider the value of bacteriological examination of the aural discharge, as determining the indications for radical interference in cases of middle ear suppuration, we are confronted by the same problem as meets us in the consideration of the differential blood count.

Combining the series of cases reported previously, with those reported at the present time, we have a series of 89 cases. In 11 the discharge was either not examined or the character of the discharge was undetermined; in 10 the discharge was sterile; in 21 the discharge showed streptococcus in pure culture; in 5 pneumococcus; in 1 mixed streptococcus; in 7 mixed infection; in 10

staphylococcus; in 13 streptococcus capsulatus; in 3 pneumococcus and streptococcus; in 3 latent streptococcus; in 3 pneumococcus and staphylococcus, and in 2 colon bacillus. Out of these 89 cases, 44 came to either mastoid or radical operation. Of the series of 37 cases, previously reported, 7 came to mastoid operation. In two of these, the infection was mixed streptococcus and pneumococcus. In one the discharge was not examined. In one the infection was staphylococcus, in two streptococcus capsulatus, and in one mixed infection with streptococcus.

Of the present series of 36 cases, in 23 the mastoid operation was performed; in 11 of these there was streptococcus infection, in 3 infection of staphylococcus, in 4 the infection was mixed, in 1 case the examination was unsatisfactory. Four cases were submitted to the radical operation. No culture was made in 3 of these cases, and in 1 case the infection was recorded as "mixed infection." Of the 9 cases in which the mastoid operation was unnecessary, no culture was made in one case. Streptococcus infection was present in 4 cases, streptococcus and staphylococcus in 1, mixed infection in 2, and pneumococcus and streptococcus in 1.

Looking over the cases of mastoid operation, we find that streptococcus infection predominated in both series of cases. This would naturally lead us to expect that, given a streptococcus infection, the chances of probable mastoid involvement would be greater than where the infection was due to some other organism. As in a number of these cases, however, the infection was of a milder nature, this rule cannot be laid down as an invariable one. It should also be noted that in 9 cases in which no operation other than free incision of the drum membrane was performed, streptococcus infection was present in 4 of these cases. It is wise, therefore, for the surgeon to bear in mind simply, that given a streptococcus infection, the possibility of serious mastoid involvement is greatly increased; that such involvement, however, is certain in a doubtful case, cannot be decided upon the evidence of bacteriological examination alone. Some of the classical symptoms, indicative of mastoid involvement, must be present before, even in a doubtful case, the mastoid operation should be advised.

Much attention has been drawn recently to the virulency of streptococcus capsulatus infection. In the series of cases reported in the article already alluded to, streptococcus capsulatus was present in 10 cases, and in only two of these was the mastoid opera-

tion necessary. In the last series of cases reported, streptococcus capsulatus infection was present in three cases. Two of these cases recovered perfectly after incision of the drum membrane, while in one case a mastoid operation was necessary, followed later by the radical operation, with opening of the labyrinth.

While this communication involves such numberless figures that it may be confusing to follow it, several important points are, I believe, clearly demonstrated: First, that in no single case of simple mastoid involvement was the increased leucocytosis or the increased percentage of polymorphonuclear cells sufficient to aid the surgeon, in any way, as to the probability of mastoid involvement; second, that while a streptococcus infection of the middle ear is, without doubt, more serious in character than some of the other infections, many of these cases recover after free drainage of the middle ear has been established by a competent incision through the drum membrane; third, that while the streptococcus capsulatus infection is undoubtedly severe and insidious in character, and gives rise to few general symptoms, the frequency with which this infection can be overcome by free incision of the drum membrane is easily demonstrated by the above statistics, namely, that out of 13 cases in which streptococcus capsulatus infection was present, in only three of these was the mastoid operation necessary. I might add that all of these 13 cases occurred in private practice, and that the patients have been under personal observation for periods varying from 3 to 18 months, so that the possibility of any unrecognized extension, due to infection, is absolutely impossible.

It would seem that the involvement of the soft tissues, either of the neck, the brain substance itself, or of the lateral sinus, would be indicated by a rapid rise in the polymorphonuclear percentage, together with an increased leucocytosis.

Quoting again from my previous article, in two cases of otitic meningitis, in neither of these cases, excepting just before death, did the blood count give the slightest hint as to the involvement of the intracranial structures. In a case of cerebral abscess, previously recorded, and in one case of sinus thrombosis, also previously alluded to, an increase in the polymorphonuclear percentage, together with an increase in the number of leucocytes present, was of aid to the surgeon. I believe, therefore, that we may safely say that an increased polymorphonuclear percentage and an increase in the leucocyte count, is of absolutely no value in determin-

ing the presence of pus in the mastoid in doubtful cases:—that where these variations from the normal blood count occur in aural cases, we must look for one of three conditions: 1st, some visceral lesion; 2nd, some involvement of the soft tissues in the immediate vicinity of the wound, and, 3rd, for some involvement of the intracranial structures, either of the brain substance or of the lateral sinus. It should be particularly borne in mind, as is clearly demonstrated by the statistics, that in certain instances, a thrombosis of the lateral sinus may occur, in which the polymorphonuclear count will not rise above 80 per cent, and in which the leucocytosis will not be greatly increased. The differential blood count and bacteriological examination of the aural discharge is certainly indicated in every case of middle ear suppuration, although the actual amount of information afforded to the surgeon is certainly problematical. The surgeon must still rely on clinical signs of involvement of the mastoid and of the intracranial structures, and must put the bacteriological examination of the discharge and the differential blood count in a secondary place, in arriving at his diagnosis.

DISCUSSION.

Dr. Wendell C. Phillips, of New York City, agreed with Dr. Dench that one should not rely upon the blood count and bacterial examination in making a diagnosis, to the exclusion of the clinical manifestations. They were valuable adjuncts and as such should be given full consideration. He emphasized the importance of having the blood and bacteriological examination made by experts, also that the examination of pus should not stop with the study of slides, but cultures should be made. This was done at the Manhattan Eye and Ear Hospital, the effect being to get as nearly pure cultures as possible. It was interesting to note in what a large proportion of pure cultures the infection was streptococcic. This bore out the careful experiments made by Libman at Mt. Sinai Hospital, in which he found that pure culture mastoid and middle ear suppuration is almost invariably streptococci infection. In the light of present knowledge, he considered them as valuable corroborative clinical evidence.

Dr. S. MacCuen Smith, of Philadelphia, said that much as he valued the differential blood count and bacterial examination of

the blood, he believed them to be secondary in importance to clinical experience. In other words, at the present time and in the recent past the tendency in aural practice has been to place too much stress on a laboratory diagnosis, but this, in his opinion, could never take the place of that riper judgment that can be acquired only by an extensive clinical experience. He therefore agreed entirely with the conclusions presented in Dr. Dench's valuable paper

Dr. Henry O. Reik, of Baltimore, Md., took exception to the too broad generalization by Dr. Dench from his experience with blood counts. He agreed with him as to the secretions from the ear but thought the blood count would be of value in many instances were it was a question whether or not a case of simple mastoiditis should be subjected to operation. The diagnosis might be made without the blood count, yet such a count might play an important part in the decision regarding operation. He cited two cases in point. In one case his associates had taken the ground that the condition was due to typhoid fever, despite the fact that the child had middle ear infection and mastoid tenderness. In typhoid fever there is leucopenia, whereas in this case marked leucocytosis could be demonstrated. Operations showed clearly the wisdom of the decision based upon the blood count.

Dr. Dench, in closing the discussion, said that where the smear was unsatisfactory he made a culture. If however, the smear showed only the streptococcus a culture would be of little value. On the other hand, if the pneumococcus was found in the smear a culture would show the streptococcus. Referring to Dr. Haskin's remarks, he called attention to the fact that the report of Dr. Libman had reference to whether the lateral sinus should be opened in doubtful cases. The presence of the streptococcus in the blood is indicative of general pyemia and consequently was not under consideration in this paper. As to the blood count being a determining factor, as suggested by Dr. Reik, in not one of forty-six cases reported had it been of any assistance. It had proved of no value even in a case of extensive mastoid destruction, where the entire mastoid was absolutely broken down and there was a large epidural abscess.

PAPER:

NEUROSES OF THE NOSE.

CHARLES PREVOST GRAYSON, M. D.

I have the somewhat uncomfortable feeling that you will discover within the next few minutes that you have been made the victims of misplaced confidence on the part of your committee who did me the honor to invite me to present this paper. I shall make no attempt to be ponderously scientific, nor have I any intention of yielding to the temptation that this opportunity offers to add something new and startling to the already formidable list of reflex nerve disturbances for which the nose has been held responsible. On the contrary, I am very much of the opinion that before any further addition is made to this list, the foundation upon which it rests should be critically examined to see if it be secure enough to support even the strain to which it has already been subjected. I am conscious that there is in this statement at least a suggestion of doubt on my part as to whether the nose is really accountable for all those ills of reflex nature that have been attributed to it. I know that whenever it has been charged with any offense of this kind more or less evidence has been forthcoming to substantiate the charge, but, although I do not venture to say in the majority, yet in many cases at least this evidence has been of a purely circumstantial character and has seldom been sufficient to induce me as one of the large jury of rhinologists to concur in a verdict of "guilty." In almost if not all of them the "neurotic temperament" has formed a more or less conspicuous background to the various manifestations of nerve irritation, and the vagaries of this irresponsible temperament are unfortunately apt to be as misleading as they are notorious. In the *absence* of this powerful contributing factor, I should regard the prompt and permanent cure of an asthma, for instance, by the removal of a septal spur as forming a *prima facie* proof of nasal guilt, but whenever its presence can be demon-

strated or even fairly suspected, the possible indirect and psychic effect of the operation would lead my conscience to decline any greater responsibility than would be involved in rendering against the nose the shifty Scotch verdict of "not proven." In the face, therefore, of the adverse criticism that I may perchance draw upon my head, I say without equivocation that to my mind whenever this troublesome temperament forms one of the premises in any proposition of a pathological nature, it vitiates and lays open to suspicion and dispute any positive conclusion that may be drawn. It is because of this embarrassing uncertainty that must always confront one in that class of nasal neuroses in which the nose is presumed to be the excitant of a reflex disturbance, that I find much greater satisfaction in dealing with that other class in which the nose itself is the site of the disturbance of innervation, that class in other words in which symptomatically the nose is the victim rather than the aggressor. The clinical features of these affections are so distinctive, so almost unique, that it is probably extremely seldom that we encounter any difficulty in the matter of diagnosis. How fortunate it would be, both for us and our patients, if our pathway to a complete understanding of the pathogenesis were as free from obstacles! I shall not occupy your time with any consideration of the functional derangements of the olfactory nerve. Ordinarily, these are of comparatively slight clinical interest and importance. It is rather to the vaso-motor neuroses as they are exemplified in the nose that I would venture to ask your attention, and I can clearly foresee the shudder with which you will suspect me of an intention to rake over the ashes of past discussions of hay fever. But this is not my principal purpose. My chief wish is to assist, simply as a mark of respect, at the obsequies of the latest "specific" for the treatment of that disease. I am not privileged to sit with the mourners, for almost from the moment of its birth I anticipated, predicted, and was prepared for its early death. And yet, I wish to lay a flower upon its grave because "pollantin" was conceived in a scientific spirit, the methods by which it was made were equally scientific and the reasoning that led to its production was, so far as it went, perfectly sound. But it went neither far enough nor deep enough. If it had, it is highly probable that no claim of specific virtue would have been made for the remedy. If the etiology of hay fever comprised no more than two factors—a predisposing

idiosyncrasy and an atmospheric excitant—there would have been nothing illogical in the expectation that "pollantin" might take its place beside the antitoxin of diphtheria, but when we recognize the existence and fully appreciate the nature of the systemic condition that, it seems to me, is more or less clearly in evidence in every case of this disease, we will not be surprised at the percentage of cases that have been reported as but slightly improved or absolutely unimproved. For many years I have believed that a fundamental disturbance of the nutritive processes, of normal metabolism if you please, was responsible for the essential nervous element in the causation of hay fever. So long as we have a blood current contaminated with a number of toxic materials, the products presumably of sub-oxidation, so long are we almost certain to have nerve centres that are poorly nourished, over-sensitive to disturbing influences, and of reduced power to neutralize the effects of such influences. It seems to be, at least in hay fever, the vaso-motor centres that are earliest and most markedly affected, and when we have added to this not only the idosyncrasy, but possibly also a long precedent intra-nasal lesion that has helped to make the pituitary mucous membrane particularly intolerant of any form of irritant, we have but to await the floating of some variety of pollen or other obnoxious material within the nostrils to witness the speedy evolution of the symptom complex of this disease. If there is one fact connected with hay fever that, to my mind, throws a stronger light upon its remote causation than any other, it is that the affection is pre-eminently one of that class of people who lead the strenuous rather than the simple life. Why is it, may I ask, that these people have an almost exclusive monopoly of what we are pleased to term idiosyncrasies? Why is it that the laboring class is practically exempt from this species of affliction? Is it not because of the facts that its members go to bed with the chickens and rise with the lark, that they have their three frugal meals each day on the stroke of the clock, that, plain as it is, their supply of food is more often inadequate to even satisfy their appetites than to satiate them, that each day's labor involves wholesome muscle work that induces wholesome fatigue and brings dreamless sleep, and that, finally, their brains and nerves know nothing of emotional strain, of vaulting ambition, of crushing disappointment, or of sleep destroying passions of any kind? Is it not possible, think you, that if, instead of fulfilling

her natural destiny, bare-footed, short-skirted, uncorseted Maud Muller had married the Judge, if she had flung aside her rake, had abandoned the cows that she had milked, the churn that had helped to make her broad of shoulder and deep of chest, and if she had substituted for these all the nerve shattering follies and vices of the modern society woman—Heaven forbid that I should attempt to enumerate them!—might there not have come a time and a condition, I ask you, when even the dim memory of raking hay would have sufficed to throw her into a paroxysm of sneezing? It may seem scarcely worth while to indulge in speculations concerning the nature of idiosyncrasies, but I have often thought that if we, as we all of us do, have certain conscious likes and dislikes, is it not possible that we have others, also, that are subconscious? Cannot our respective mucous membranes, for instance, owing to some individual peculiarity of structure or innervation, have certain antipathies of their own of which we may be wholly unconscious until lowered vitality, increased peripheral nerve irritability and lessened central control permit them to emerge from the obscurity of the back of the stage into the glare of the limelight at the front? I have fancied that we may either inherit or acquire in some way the seeds of these idiosyncrasies, but that they may lie fallow and inert until they are fertilized by and spring into life and offensive activity from a soil of poisoned and debilitated nerves.

What a sublimely sanguine nature it must be, then, that still clings to the hope that a remedy of specific power may yet be discovered for hay fever! When a man so qualified by exceptional experience and unimpeachable judgment as Holbrook Curtis commits himself to the statement that "hay fever is a disorder amenable to no specific treatment," it seems to me that there is nothing left for the rest of us but to accept his opinion and to finally abandon that fatuous hope to which I have just alluded. And so, upon the tributary wreath that I cast upon the sod that covers "pollantin" I would write the inscription "Sic semper specifics." One more word relative to the treatment of this disease and I will take my leave of it. With no lack of respect for the profound laboratory studies that have dealt with the secretions of the hay fever sufferer, I fail to find in their results anything that is of genuinely practical value to us. It may be of some academic interest to know that the sweat contains an excess of something,

that the tears that flow so abundantly during the attack contain too much or too little of something else, or that the saliva, perhaps, shows the presence of some material that is foreign to it, but of infinitely greater importance is it to remember and to remove the basic nutritive fault that is accountable for such alterations. This is a matter I think, to which the maxim "Immanente causa non cessabit effectus" is clearly applicable. Surely the drug treatment of hay fever, from a curative point of view, is utterly irrational and fallacious. Nothing short of a drastic and radical reform of each patient's personal hygiene can effect and maintain the normality of the body tissues and secretions. I speak feelingly because as an ex-victim of the disease of some years' standing, I am confident that if I were to renounce systematic exercise and to return to indolence and the flesh-pots, the coming of August would find me once more a refugee in some mountainous region or upon the rag-weedless bosom of the Atlantic. If I were asked, therefore, in which of the two places, the laboratory or the gymnasium, a cure for this disease were the more likely to be found, you probably know what my answer would be. To put it in a nut-shell, it is my belief that if a man takes care of his muscles, with *all* that this implies, his nerves and vaso-motor centres will take care of themselves.

Another nasal neurosis very closely allied to hay fever, in fact, almost identical with it symptomatically, is that termed "rhinorrhoea spasmodica" or "paroxysmal sneezing." Pathologically, it seems to be hay fever minus the latter's idiosyncrasy concerning external irritants. The irritants of internal production are no doubt precisely the same in the two affections, and in both, also, the secondary nervous element is equally in evidence. This latter, however, in rhinorrhoea spasmodica is more often temporary than temperamental. In other words, the disturbance of vaso-motor equilibrium is the result in most of these cases of some accidental and transient stress of an emotional nature, and it is this which may partially account for the absence of any periodicity in connection with repeated attacks of this disturbance. In illustration of this statement, I may be permitted to briefly refer to three cases that I have seen within the past two months, in none of whom could any evidence be found that would justify their being regarded as either hereditary or habitual neurotics, and none of whom had ever had a previous attack of this nature. The first

was a man of middle age who was deeply involved in the stock market last Fall and who throughout the Winter was compelled, for want of margin, to let his holdings go one by one until, with the coming of Spring, he was as much a nervous as a financial bankrupt. The second, a woman who, during the several weeks of its closely preceding critical illness, could scarcely be dragged from the bedside of her child. And finally, a trained nurse of rather frail physique who, for an unbroken period of six weeks, had been vigilantly watching a patient possessed of a suicidal mania. The pathogenic fact that was common to these three cases was a prolonged and flagrant violation of those laws of personal hygiene relating to food, sleep and exercise, and the result, common also to all of them, was a most pronounced impairment of nutrition and of nervous vitality. Associated with the autotoxemia and nutritive failure were both unusual phosphatic waste and defective elimination. Although in each of them there was only such intranasal abnormality as would, under ordinary circumstances, have produced no discomfort, yet aided and abetted by the foregoing conditions it proved quite sufficient to induce indignant Nature to select the nose as the medium for her protest.

In conclusion, gentlemen, I am going to recall to your attention a third neurosis of the nose that has its origin in disorderly sexual functions. In the first place, we are all familiar with the fact that during the menstrual period not only is any coincident affection of the female nose apt to be aggravated, but even if it be normal it is often apt to reflect to some extent the ovarian excitement. It was perhaps his knowledge of this fact that led Doctor ——— to seek a cure for dysmenorrhoea through the cauterization of those areas in the nose that showed most plainly the secondary hyperemia and hyperesthesia. Shortly after the publication of his observations probably many of us through the kindness of our obstetric and gynecologic friends had opportunities for testing his conclusions, but I must confess that the results of my own series of cases developed little more than the opinion on my part that while in women functional derangement of the pelvic viscera was capable of producing more or less disturbance of the neurovascular mechanism of the nasal mucosa, yet that the control of this latter would have little or no appreciable effect upon the former. Always excepting, of course, the highly neurotic woman, and upon her there is scarcely anything in the fields of medicine,

surgery, Christian science or osteopathy that may not produce the most extraordinary cures. In close connection with this, it is just barely possible that some few of you may have a dim recollection of a paper that I read at the semi-centennial meeting of the American Medical Association in 1897, concerning the influence of sexual excitement upon the intranasal circulation. The two cases that I then reported have since been followed by many others, and, could I conquer my aversion to hair-splitting distinctions in the sub-classification of disease types, I would venture to suggest to the author of the next text-book on diseases of the nose that he devote a section to "The Rhinitis of the Newly-Married," or, perhaps, "Honeymoon Rhinitis" would be shorter and more euphuistic. I have not introduced this subject in order to discuss its treatment, because as a consistent bachelor I could scarcely do otherwise than advocate preventive rather than curative measures, but I refer to it once more simply to express the opinion that the cause of this neurosis of the nose is as frequently overlooked or viewed with the same indulgent indifference to-day as it was ten years ago.

DISCUSSION.

DR. B. R. SHURLEY, of Detroit, Mich., had made an effort to determine how much of reflex disease of the nose could be referred to as general neurosis. He had not been able to find a single instance of the ordinary neuroses which was not accompanied by some other symptom of general neurosis. Careful examination such as the internist would make revealed other neuroses in almost every individual, and while the rhinologist was treating such an individual for the nasal condition, the patient was going the rounds of the various specalists in other fields. Innumerable symptoms were complained of by these patients and while there was a method of treatment, each individual case must be studied. By the use of the valerianates, asafetida, by dealing with the predominating neurosis, and by treating these patients as tuberculous individuals are treated in the early stages of the disease, the interval between the nerve storms can be generally lengthened. These cases presented the distinct pathology of nerve-center disease. Some years ago, he had the opportunity of examining histologically the brain cells of a carrier pigeon after a long journey. Protoplasm of the nerve cells showed a distinct histogenic change. The granules were

all used up. A distinct difference could be seen between the protoplasm of the pigeon which had been resting and that which had been working, and the same applies to the human tissue.

Dr. Lewis A. Coffin, of New York City, asked the reader of the paper just what he meant by nasal neuroses and reflexes, and where he considered the sensory nerve as having its origin. There seemed to be many things involved in this question of nasal neuroses not understood as yet. It was well known that some people sneezed when they looked at the sun. A physician whom he knew sneezed every morning until he had an action from the bowels. These, of course, he did not consider nasal reflexes but they were not well differentiated. He did not agree with what had been said about the strenuous life in this connection, for children who had not become strenuous had hay fever, as had also boys who were athletic. He was thoroughly opposed to operating in the nose for this class of cases until a more careful study of the case had been made.

Dr. J. A. Stucky, of Lexington, Ky., called attention to the fact that the conditions under discussion are not seen in laboring men, nor are they seen in the negroes of the South; neither are they seen in the classes referred to by Dr. Coffin except in such individuals as are in the habit of taking large quantities of sweets and alcoholics. It was, in the majority of instances, a question of faulty intestinal metabolism. Neuroses of the nose were found in the over-fed and under-worked. In over four hundred observations he had found an excess of indican, showing that the intestinal function was not properly exercised. He recommended a return to the "simple life."

Dr. Coffin added that he had known three cases of asthma, one of which was cured by Christian Science, one by having the nose thoroughly cleared out and another by having the eyes treated. Still another case was cured by having a piece of coal removed from the ear.

Dr. Henry O. Reik, of Baltimore, Md., said that it mattered not whether Pollantin ever relieved a case of hay fever, the fact remained that Dunbar was the only man who ever approached the study of that disease in a scientific spirit or offered a reasonable scientific theory to explain it. He felt that we owed him thanks,

not ridicule, for his labors and it was high time we devoted ourselves to a scientific investigation of these nasal neuroses instead of considering them as subjects for a joke.

Dr. Stucky had spoken of the hay fever patient as one who was "over-fed and under-worked." He felt that he was the antithesis of that for he, surely, was overworked if not underfed, and yet one of the miserable victims of the disease, which might account for his not getting as much pleasure out of Dr. Grayson's paper as most of the Fellows seemed to derive.

PAPER:

REPORT OF A CASE OF PROBABLE PERSISTENT ANGIO-NEUROTIC EDEMA OF THE LARYNX AND SOFT PALATE.

T. H. HALSTED, M. D.

At the meeting of this Society held in New York City one year ago, I presented for examination and diagnosis a case which excited a good deal of interest among those of the Fellows who examined the patient. The case was that of a young woman with an obscure infiltration of the soft palate and larynx, and will be recalled by some present. While in New York she was also examined, at his office, by Dr. Prince Morrow, the distinguished dermatologist and syphilologist, who was as uncertain as to the nature of the disease as were the laryngologists who examined her. I reported the case as possibly one of persistent angio-neurotic edema, and in reporting the progress of the case to-day I do so under the title of "Probable Angio-Neurotic Edema," showing the continuing uncertainty of my opinion.

Syphilis, tuberculosis, or lupus was the opinion of perhaps the majority, while a few others thought it might be the result of some undiscovered circulatory or lymphatic disturbance, or related to some disease or disturbance of some one of the internal secre-

tory organs. Without exception, no Fellow felt confident of his opinion. Professor Killian, who examined the case with great care, thought it probably syphilitic.

Miss E. V., age 30 years, bookkeeper, living in Cortland, N. Y., consulted me on June 15, 1905, because of a thickness and fulness in the throat, dyspnea on exertion, post-nasal and nasal discharge, excoriation of left nostril, and frontal pain. These symptoms had followed a quinsy which occurred four months before, and which had not entirely cleared up after the abscess was lanced. On examination there was an apparent collection of fluid in the right anterior pillar of the fauces, the velum palati was thickened, the uvula swollen, and arytenoids were pale and swollen. There was some dry crusting over the middle turbinate and a little pus in the right middle meatus, coming apparently from the anterior ethmoid. Transillumination was negative. Nothing adventitious in the chest. No sputum. Temperature normal. She had lost ten pounds in weight during the past three months, and was on her way to the Adirondacks through the advice of her physician. I incised the swollen anterior pillar and let out a few drops of pus. I suspected tuberculous disease of the larynx and soft palate. I did not see her again for five months, when she returned and presented almost exactly the same condition as on my first examination. In the meantime she had hawked out a flat thin piece of bone, as large as the thumb nail, which had come from either the nasal septum or the ethmoid.

There was a leathery, pale infiltration of the uvula and soft palate, both arytenoids were swollen, pale pink, edematous in appearance, but leathery to the probe, pyriform in shape, the epiglottis upper surface infiltrated. No ulceration, no pain, no dysphagia, but dyspnea on slight exertion. The septum was superficially ulcerated and crusted on both sides, no bare bone detected anywhere, some ethmoidal suppuration on right side, the nose at the tip reddened, and some excoriation at nostrils. Repeated examinations of sputum were negative. Nasal scrapings were negative. Temperature taken several times a day, and for many days, was normal. Pulse normal. General health good, no loss of weight. Repeated urinary examinations by competent men were always normal. Gastro-intestinal tract was normal.

Family History.—I have never been able to obtain a very satisfactory family history, never having seen any other of the family than the patient. Her own physicians in Cortland, first Dr. Carpenter and latterly Dr. Dana, both of whom have been greatly interested in the case, and well acquainted with the family, have never discovered any history or evidence of tuberculosis or syphilis or other constitutional dyscrasia in the family. The mother is living and well. Father died of pneumonia a month ago.

Previous History.—At six years of age patient had scarlet fever, followed by enlarged cervical glands, which finally subsided without suppuration, but an ulcer of the lip extending into the nostril persisted for four months, finally healing without scars. No other illness of note until she was 18 years of age, when she suffered a great deal from chilblains, and about this time a small swelling occurred over one heel. This suppurated, the bone was curetted, and after several months the wound healed, leaving a scar. This was said to be scrofulous.

The patient now enjoyed good general health, without any local disease anywhere, for 12 years, i. e., until she was 30 years of age, when she first came under my observation with the condition as described at the beginning of the report.

The condition then, three years ago, was a distinctly leathery, pale, swollen infiltration of the uvula and soft palate, especially of the right anterior pillar, epiglottis pinkish, edematous on its anterior surface and margins, a pale edematous pyriform swelling of both arytenoids, partially obscuring the vocal cords, which, however, were normal. In the nose was a crusting or scabbing of the septal mucous membrane, a fissure at nostril and some redness of tip. Voice was clear, slight dyspnea on exertion, with at times severe exacerbations, requiring sitting up in bed at night, these exacerbations lasting a few days and subsiding, no pain whatever either on swallowing or referred to throat or ear, no cough, no expectoration, no pyrexia. General health good. Heart, lungs, kidneys, gastro-intestinal tract all apparently normal.

Such was the condition when she came under my care three years ago. It continued the same until she appeared before this Society one year ago, with the addition, however, that a

month prior to coming to the meeting she had an attack of what her physician diagnosed as facial erysipelas, differing from erysipelas in that a thickening of the lip and of the skin over the cheeks persisted after the active disease had subsided, and was present when I presented her to the Society.

After first coming to me, three years ago, repeated examinations of the sputum and nasal secretions and scrapings were made for tubercle bacilli, but none were ever found. Temperature was always normal. She was early placed on anti-syphilitic treat ment extending at intervals over long periods of months, iodine in its various forms, the iodide of potassium up to 200 grains a day, and mercury in various forms, by mouth and inunction. Various applications were made locally. Nothing had any appreciable effect, the disease not progressing but not subsiding. She was repeatedly seen with me from the beginning by Dr. H. C. Baum, a dermatologist and syphilologist, and it was at his suggestion that I had her seen by Dr. Prince Morrow, of New York. Dr. Morrow could find no evidence of any inherited or acquired syphilis, and thought the skin infiltration of the lip and face were not erysipelatous, but would not make a diagnosis. He suggested, however, the use of salicylate of mercury hypodermatically, a suggestion also of some of the laryngologists who examined her. Other suggestions made at this meeting were excision of a piece of the infiltrated tissue for microscopic examination, especially with reference to lupus, and the tuberculin test. I have not thought it wise or safe for the patient to make use of either of these two measures, desirable as they might be from a diagnostic point of view.

A course of salicylate of mercury has been given, consisting of twenty deep hypodermic injections. During the past year there has been no material change in the local condition of the uvula, soft palate and larynx. The same pale, leathery, edematous infiltration which you saw a year ago persists, although the swelling seems to be slightly less, and dyspnea is seldom complained of, and acute exacerbations of difficult breathing do not occur. The nasal condition is unchanged. In the face, however, there has been a very decided degree of activity. During the year she has had five or six attacks of what her physician at home, Dr. Dana, at first regarded as facial erysipelas, but he has now

given up that opinion. Dr. Baum and I have always regarded them as angio-neurotic edemas. Without any known or apparent cause, there comes on suddenly great redness and swelling of the face, usually of one cheek, often of both, sometimes involving the dorsum of the nose and lip, no pain, but a burning sensation. This usually reaches its height in a few minutes or hours, sometimes extends, often does not. Once it occurred immediately following, in my office, a salicylate of mercury injection, so that the face became greatly swollen, cheeks deep red, one eye nearly closed, all coming on in a few minutes. Sometimes the temperature reaches 104° F. during the attacks, at other times temperature is not elevated. When these facial swellings first occurred she stayed in bed, and the disease was treated as erysipelas; of late she pays no attention to them but continues at her work as embroiderer, and finds herself recovering as quickly while up and attending to her work as formerly when she remained in bed. Since her first facial attack, more than a year ago, there has been constantly and persistently some swelling, brawny, and rather leathery, of the skin of the upper lip, right cheek, and rather edematous of the loose tissue under the lower eyelids of both sides, the so-called attacks being apparently but acute exacerbations of what I believe is a persistent angio-neurotic edema involving the face. In other words, I believe that the scene of activity has shifted from the pharynx and larynx to the skin of the face, while still persisting in these first locations. I feel certain that there is some underlying cause, constitutional or local, some dyscrasia, or some local source of toxic absorption, the nature and source of which we do not know.

PAPER:

DISEASES OF THE NASAL ACCESSORY SINUSES AND THEIR RELATION TO PATHOLOGICAL CHANGES OF THE EYE AND ORBIT.

CHRISTIAN R. HOLMES, M. D.

This subject has become so extensive that in a paper like this, one must confine himself to a brief resumé of the most important facts. Although the anatomy of the nasal accessory sinuses of man has been well known for about thirty years, thanks to the indefatigable work of Zuckerkandl and others, and the comparative anatomy of these cavities has been worked out for some of the higher animals, their functional uses are still a subject of speculation for the physiologists who have evolved some most ingenious and interesting theories as to their various functions and reason for their existence. But a knowledge of the diseases of this part of the upper air tract is a matter only of the pathological yesterday.

This latter accomplishment is due to the rhinologists, whose daily contact with the nose and throat gradually impressed upon them the dangerous character of the inflammations of which they are frequently the seat.

Cases such as are now familiar to all of you, as lately as ten years ago were deemed to be in the province of the physician, the ophthalmologist or the dentist, and it was only when the rhinologist, with his special knowledge of the anatomy of the head and neck and his abundant clinical experience of the diseases of this region, began a systematic study of the accessory cavities for the purpose of working out a safe and intelligent surgical anatomy, that it was possible to present to the scientific world a complete exposition of the etiology, symptomatology, pathological anatomy, bacteriology and operative treatment of the diseases of the accessory sinuses of the nose. Their location, shape, structure and relation to important adjacent or surrounding structures, makes treat-

ment or surgical interference often both difficult and dangerous. Their walls are frequently only of paper thickness, and in direct contact with vital structures. Under such conditions we are frequently unable to remove all necrosed bone. Take, for example, the sphenoid cavity; its walls are always thin, and at times dehiscent in the region of the optic nerve and cavernous sinus—we now know from postmortem reports that the walls are often necrotic—and while you may make a free opening in, or even remove all of the anterior wall, you cannot possibly, with safety, remove the other necrotic walls of this sinus—but must trust to the natural process of exfoliation and repair. Sometimes we have in any or all of these cavities septa and diverticula or irregularly projecting cells, that it is almost impossible to reach, unless the most radical operation is resorted to; and even then, the field is not always clear, especially in the region of the sphenoid. The natural openings into the cavities are so located, that in disease, drainage is greatly interfered with; and especially is this true in the case of the antrum of Highmore and sphenoid, where they are near the roof instead of the floor. The outlets from the other cavities are more favorably placed, but polypi or hypertrophied turbinals can most effectually close them off.

The manner in which infection enters these cavities in the vast majority of cases is through the natural openings when the patient is suffering from an acute or chronic infection of the Schneiderian membrane.

Dr. Robert Falcone recently tested the correctness of the theory of Harke and Hajeck, and proved that infected material may be forced into the sinuses during violent sneezing or blowing of the nose. These experiments were made on dogs; whether they will hold good to the same extent in man it is difficult to tell, but we do know that during the act of vomiting, some of the contents of the stomach have been forced into the accessory sinuses.

Infection, no doubt, sometimes extends from the nares to the sinuses through the blood and lymph vessels of the contiguously inflamed tissue.—swelling of the mucous membrane in the nose may occlude the natural openings which is soon followed by an absorption of the contained air, creating a vacuum. This leads to transudation of serous fluid into the cavity as we find it in the middle ear. This fluid forms a favorable medium for the development of pathogenic bacteria should any be present or gain entrance later.

Carious teeth play a somewhat important role in the development of antral suppuration. While the dentists and many rhinologists formerly attributed nearly all cases of antral infection to dental disease, it is now generally accepted that from five to ten per cent is more nearly correct. From the specimens that I have examined, I feel convinced that the percentage must be small.

It must not be forgotten that the cavities having no connection with the teeth are just as liable, if not more so, to be infected, than is the antrum of Highmore.

Harke, in 37 postmortem examinations of children dying of diphtheria, whooping cough, scarlet fever, measles and chicken-pox, in every case found suppuration in one or more of the accessory cavities, the maxillary antrum being the most frequently affected. While children are frequently attacked, fewer cases come to operation than among adults, because, usually, their recuperative powers are greater than those of adults. The most frequent cause is undoubtedly influenza.

Dr. Darling, Pathologist to the Ancon Hospital, Isthmus of Panama, conducted an investigation to determine the relation of inflammations of the accessory nasal sinuses to pneumococcus infections. The sinuses were examined with regard to this point in 52 autopsies, 37 of which were pneumococcus infections, as follows: Lobar pneumonia, 22; acute pericarditis, 1; acute meningitis, 9; pneumococcus septicemia, 5. He found that 92 per cent of all pneumococcus infections coming to autopsy showed in a very marked degree more or less typical pneumococcus inflammation of one or more of the nasal accessory sinuses. The inflammation was generally intense. It was fibro-purulent in character. Pneumococci were always present, and in numbers dependent on the duration of the process. A point of great importance was that the age of the sinus infection had been appreciably greater than that of the lung of meningeal lesion. Ninety-one per cent of the lobar pneumonia cases showed a sinusitis. All cases of acute pneumococcus meningitis presented an inflammation of one or more of the sinuses, and in every one the middle ears and mastoid cells were normal. In the pneumococcus septicemia group 80 per cent were found to be associated with a sinusitis. In the case of acute pericarditis all the sinuses were involved. In fifteen autopsies of other than pneumococcus infections 28 per cent had an acute inflammation of the sinuses. Darling believes "that the portal entry of the

pneumococcus is in most instances an accessory nasal sinus, the mucous membrane of which is probably fitted for the reception of the pneumococcus by an antecedent influenza or rhinitis."

Erysipelas has been regarded as a cause, but it can now be positively asserted that it is *secondary to sinus disease*. In a study of this subject which I undertook about a year ago, I found a vast mass of recorded cases, the details of which proved conclusively that attacks of facial erysipelas invariably arise from an infection due either to an increased virulence of the streptococci which inhabit the diseased nasal chambers, or to a reduction of the vital powers of the patient inviting an infection by these micro-organisms, or, more probably, a combination of these two active factors in disease. Tuberculosis may, by ulceration and necrosis, extend from the nares into any of the sinuses. I have seen but few such cases, and cannot help wondering if they are as frequent as some would have us suppose. On the other hand, syphilis is frequent, but, as a rule, I have found that it yields readily to vigorous internal treatment. Malignant tumors may invade or develop within the cavities. It is needless to state that an early and correct diagnosis is of vital importance. If the disease is at all extensive, the prognosis is always very grave. It is remarkable to what an extent these growths may cause displacement of the contents of the orbit without interfering with vision, or even causing diplopia. This is, of course, explained by the absence of inflammation, and the slowness of the growth which enables the parts to adjust themselves to existing conditions. Individually I have come to the conclusion not to use the knife hereafter on malignant tumors in this region—it is so hopeless. I know of only one method that gives any hope at all, and I intend to use it in all future cases wherever possible. I refer to the cataphoric treatment of malignant growths by the Belton Massey method. Either use his hollow gold electrodes filled with metallic mercury, or the zinc electrode, amalgamated at the moment of using with sulphuric acid and mercury, is the proper method. By using from 400 to 1,000 milliamperes of current under a pressure of from 50 to 75 volts, most remarkable results can be obtained. Massey's explanation of the action of this cataphoric sterilization is as follows: "By the electrical conversion of metallic mercury injected into the growth into a soluble oxychloride, which is then diffused in all directions through the growth, the protoplasm of the cells, germs and all, is converted into an al-

buminate of mercury, rendering the dead mass thus formed aseptic and odorless until it drops off, leaving a clean wound to be healed by granulations. But the method does not stop its usefulness at the edges of the slough thus formed. This area of destruction, which should be coterminous with the supposed edges of the growth, is surrounded by a sterilized zone of varying extent, within which the diminishing density of the radiated chemical leads to the death of the lowly organized cancer cells only, the normal tissues being merely stimulated to a greater physiological resistance."

I used it successfully seven years ago in a case of sarcoma involving the tonsil and base of the tongue, and not as yet reported. Recently I have used it again in a case of sarcoma of the hard and soft palate the size of half a small orange, under general anesthetic of about one hour's duration, leaving the whole tumor a grayish white, shrunken, coagulated mass. This method is, of course, not applicable in a cavity enclosed by bone until you have surgically exposed the growth.

Non-malignant growths, such as fibromas, generally originate in the fibrous tissues beneath the mucous membranes of the naso-pharynx, and invade the sinuses or cranial cavity secondarily after the bony walls have been absorbed by pressure. They frequently set up a purulent sinusitis, a condition that adds to the danger of their removal. Such a case I reported last year in the Ohio Medical Journal. The growth was a dense fibroma arising from the fibrous tissues on the anterior surface of the sphenoid, and had eroded the bony structures in its neighborhood until it had invaded both frontal sinuses, the ethmoid labyrinth, the right maxillary antrum, and the anterior cerebral fossa.

A purulent ostitis—for it was impossible to localize the process by an anatomical regional name, it was so diffused—complicated the case and made it one of that dangerous class to which I have referred, and a fatal ending followed five days after the operative removal of the growth.

The membranous lining of the sinuses may be regarded as a modified continuation of the Schneiderian membrane. Lothrop states that the mucous membrane which lines the frontal sinus "does not differ in essential characteristics from that found in the other accessory nasal cavities. In general, it is somewhat thinner than that found in the antrum of Highmore, and more easily detached

from the bony wall of the sinus. In section it presents the following layers: Facing the cavity of the sinus is a layer of ciliated columnar epithelium between the cells of which are interspersed a variable number of goblet-cells. The cilia produce a current toward the ostium frontale. Underneath the epithelium is a layer of loose connective tissue cells between the meshes of which are glands and round cells. This layer is capable of enormous increase in thickness, in consequence of inflammatory processes, by the addition of the serous element and leucocytes from the blood. The cavity of small sinuses may thereby be obliterated, and the consequent pressure gives rise to excruciating pain. The lining of the frontal sinus contains fewer glands than that of the antrum, and their distribution over the surface is unequal. Underneath this loose layer we come to a rather dense basement layer, composed of compact fibrous connective tissue cells. This is next to the bone and serves as a periosteum. It is easily detached, except in the vicinity of the ostium frontale, where it is continuous, directly or indirectly, as the case may be, with the mucous membrane of the nasal cavity. Delicate vessels passing between the membrane and the sinus wall, help retain these structures in approximation."

Twenty years ago dacryocystitis was a very common affection, and many of us can remember seeing in clinic and private offices patients sitting about with silver probes projecting from their nasal ducts. I venture to say that you saw 20 of them to one that you see now. The oculists of those days were treating the symptoms instead of the disease, and continued to do so until the rhinologists discovered that tear sac affections are nearly always due to an inflammation having its primary seat in the nose. Treatment at the lower end of the canal, instead of probing from above will, as a rule, bring quick relief in acute cases. An undiscovered purulent inflammation of the antrum may extend to the tear sac, causing purulent dacryocystitis, for which the patient seeks the advice of an oculist,—I have described such a case in Hansel & Sweet's "Diseases of the Eye." In that case I found that we had to deal with a perisacular abscess—and every accessory cavity on that side was in a state of chronic empyema—all cured by a radical operation. Cysts may spring from any cavity. The frontal sinus is, perhaps, most frequently affected. The most typical case I ever encountered is also figured in the text book quoted above. It occurred in a woman about 55 years of age. The swelling appeared long be-

fore she sought advice. The globe was enormously displaced downwards and outwards—movements of the eye were almost entirely abolished. The cornea was exposed, requiring a pad and bandage over the lids for its protection, yet vision was practically normal and ophthalmoscopic examination negative, except for a slight dilatation of the veins.—operation revealed that the cyst had eroded all the anterior portion of the os planum and the anterior ethmoid cells, the floor of the frontal and most of the dividing septum between the two frontal sinuses. The contents were of a greenish brown color, and sticky, oily consistency. Analysis and bacteriological examination showing it to be mucous and free from organisms. These cysts are always of slow development, are firm, elastic or semi-fluctuating on pressure at the center, but have a firm bony or ring-like feeling at the base. Epiphora, and in some cases, diplopia, are the only symptoms in this class of cases. The nasal examination is generally negative. The etiology of mucocele is nearly always a previous catarrh, with closure of the ostium and accumulation of mucus, the external swelling not developing till months or years after the primary catarrhal attack. Turner states "that the ostium is generally closed during the formation of the mucocele, but at the time of operation may be found open. In the latter case the contents of the cyst do not escape because they are too thick."

Osteomata are rare; they may develop in any of the cavities, or within the nose. They are usually pedunculated and of ivory hardness. They may extend into the orbit causing displacement of the globe with epiphora.

I recently removed one the size and shape of a hickory nut,—it sprang from the upper portion of the antrum of Highmore, growing into and obstructing the left naris and causing epiphora. It was removed through an extensive infra-orbital incision—a scar in this region indicating where a similar growth had been removed 18 years before—so that we may possibly regard this as a recurrent growth.

In considering the purely inflammatory conditions, we must divide them into two groups—acute and chronic, bearing in mind that the chronic varieties are liable to acute exacerbations, the pathogenic organisms becoming *for the time being very virulent.* Especially must we fear the streptococcus. It is during such pe-

riods of *increased virulence* that it behooves us to think twice before operating lest our case develop erysipelas (auto-infection) or the infection lead to venous thrombosis, meningitis, or brain abscess.

One of the most serious but infrequent results of a sinus infection is an infective thrombosis of the cavernous sinus with involvement of the contents of the orbit. One can readily see how infection or thrombosis may extend from the ethmoid vein into the ophthalmic vein and cavernous sinus, or by direct extension through the thin or dehiscent walls of the sphenoid cavity, just as we can trace the disease from the mastoid cells to the thrombosed lateral sinus.

An important point that I wish to emphasize is this—in purulent frontal, ethmoid or sphenoid cases (i. e. the cavities close to the brain) we should avoid making any minor intranasal operation shortly before the radical operation is undertaken, because even a slight operative attack in an infected nose may cause an increased virulence of bacteria. Killian says "the most difficult thing is to estimate the virulence of the bacteria." In one acute case we may operate boldly with favorable results, while in another with apparently identically the same set of conditions, so far as we are able to tell by any and all means at our command, infection develops, and the case goes on to a fatal termination. We should therefore make it a rule, where possible, to operate as the general surgeons do for appendicitis; i. e., *after* the acute symptoms have subsided, or, if it should become absolutely necessary, make only a sufficiently large opening to drain the cavity *for the time being*. We should, of course, always be specially careful when in the region of the cribiform plate—or the exposed dura—where the inner table is necrosed. Here we often find ragged, unhealthy looking adhesions formed around the opening. This is the "phagocytic wall," nature's great barrier against meningitis, *and one we must not disturb!* The violation of this rule by the over radical operator, I believe, has cost many lives.

If we note how the veins from these infected sinuses, as, for example, the ethmoids and frontal, drain into the ophthalmic vein and cavernous sinus, or even pass through the bones in contact with the membranes of the brain, or enter the brain itself—we must marvel that there are not more fatal cases.

The light that has been thrown upon the etiology of many affections of the eye and its appendages, has resulted from the joint work of the ophthalmologists and rhinologists. It is not so many years since all headaches and eye pains that did not yield to the treatment of the family physician were sent to the oculist for glasses—and, for a time, the claims of the refractionist ran riot—according to some there was nothing that properly fitted lenses and prisms would not cure, from insanity to hemorrhoids. Many were cured because they *did* need glasses—others temporarily helped by "suggestion"—but a large percentage were not helped because the seat of the trouble was in the nose and sinuses, and not in the eye. We now know that the majority of these cases can be cured or relieved by proper treatment of the upper air tract—but here again we must not let our enthusiasm claim everything, for although in some cases the symptoms may be referred to the head, the cause may be located in a distant part of the body, as was well illustrated in one of my patients, a woman of 38 years, well developed and nourished, who was under my treatment for progressive catarrhal deafness and tinnitus, with naso-pharyngeal catarrh—no sinus involvement could be demonstrated. While in my hospital she developed a violent attack of glaucoma, complicated with plastic iritis coming on the day her menstrual period began. From the history now elicited, I learned that she had during the past 18 months suffered pain and redness of the same eye at every menstrual period, which discomfort, however, generally subsided with the cessation of the flow. This was the first violent attack. A gynecological examination revealed the need of a curettment of the uterus, which was performed, and the eye became normal; and although several years have elapsed, she has never had the slightest irritation of the eye, which quickly regained perfect vision.

We have now many cases on record where partial or total blindness of one or both eyes with violent pains, has been promptly relieved by recognizing the presence of an empyema in one or more of the cavities, and instituting the proper treatment. In this class of cases it is nearly always the sphenoid cavity that is at fault, because of its intimate relation to the nerves, arteries, veins and muscles of the eye. The eye seldom causes any trouble with the nose and sinuses, but the latter very often involves the eye and orbit. It is important, therefore, that all ophthalmologists should know more about this subject in order to be able to recognize cause and effect.

St. Clair Thomson says that "many cases present themselves in the first instance to the ophthalmologist, so much do the eye symptoms induced overshadow all others."

In this class of cases we find as leading symptoms, pain, steady or intermittent, contraction of the field of vision, general asthenopic symptoms, hazy vision, tenderness of ocular muscles, inflammation of the conjunctiva and uveal tract, optic neuritis and atrophy; but this part of the subject I shall leave to one who, by his keen observations and facile pen has rendered most valuable service to our specialties—I refer to Dr. Posey, who honors me by opening the discussion on this paper.

The symptoms are indicated in the accompanying table.

Arteries and Veins.

NOSE.

Arterial Supply—Upper nasal region, anterior to the ethmoid cells, by the *ethmoid arteries.*

Artery of the Septum, a branch of the Spheno-palatine. Anastomoses anteriorly with the Descending Palatine from the Spheno-maxillary portion of the Internal Maxillary.

Posterior nasal branch of the Nasal or Spheno palatine supplies the mucous membrane of the lateral wall of the nose.

Lateralis nasi from the Facial Artery supplies the ala and dorsum of the nose and anastomoses with its fellow of the opposite side, the nasal branch of the ophthalmic, the artery of the septum and the infra-orbital.

Nasal—the distal termination of the Ophthalmic—on the face—supplies the superficial structures of the nose and the lachrymal sac, and anastomoses with the angular from the facial and the vessels of the opposite side.

Infra-orbital supplies the lachrymal sac—anastomoses with the facial and ophthalmic.

NOSE.

Veins—1—*Plexus nasalis externus*—passing forwards to the external nares.

2—*Venae ethmoidales interiores et posteriores* passing upwards into the cranial cavity and the orbit.

3—*A group passing backwards* to the soft palate and the pharynx.

4—*A group passing backwards and upwards* through the spheno-palatine foramen.

SYMPTOMS	ANTRUM OF HIGHMORE	FRONTAL SINUS.	SPHENOID CAVITY.	ETHMOID CELLS.
Pain	In teeth, in upper jaw and nose; sometimes only in frontal region. May radiate over whole side of head.	Supra-orbital weight and oppression above and behind the eye.	Frequently occipital sometimes excruciating. Lack of the eyes. Considered by some as the most characteristic. Sometimes in vertex, or in temples. Frontal. Deeply seated in ear (In 9 cases out of 42 cases with autopsy). (Mastoid opened in 3 cases and sigmoid sinus in 2 cases). Sometimes referred to other sinuses. Pain on opposite side.	Frontal. Bridge of nose.
Tenderness	Over the canine fossa and exit of the infra-orbital nerve.	Over the frontal region supra-orbital notch and above the inner canthus.	Some observers have found none, but taking all the literature together, tenderness over the eye is frequent.	Above the inner canthus.
Discharge	Can be seen covering the inferior turbinal and floor of the nose.	Can sometimes be seen coming down from the infundibulum.	Sometimes none visible to good observers. Sometimes thick pus on cavum pharyngeum. Sometimes far back in middle meatus.	Can be seen at various points in the vault,—location depending upon whether anterior or posterior cells, or both, are involved.
Swelling.	Sometimes quite marked over whole upper jaw. Sometimes completely absent.	Not infrequent above the inner canthus and root of nose. Sometimes due to external periostitis.	None external over face. Internal swelling of structures from middle turbinal up on affected side. St. C. T.'s case. Swollen purulent glands under sterno-cleido mastoid on affected side. Oedema of lids with thrombosis cavernosus.	None external—unless pus threatens to invade or invades orbit through plate of ethmoid—not infrequent in infancy.
Signs or symptoms on part of eyes	Sometimes only congestion of conjunctiva. Lachrymation. Photophobia. Rarely orbital abscess.	Occasionally direct extension to orbit and formation of orbital abscess. Corneal abscess (Fish). Erysipelas appearing about the orbit. Ocular symptoms occurring during influenza are suggestive. Glaucoma."	Pressure symptoms upon one or all of motor and sensory nerves and on vessels of orbit and globe. Papillitis or atrophy. When meningitis supervenes, unequal pupils, hazy discs, strabismus, etc. When cavernous thrombosis appears, proptosis and chemosis. Eyelids tense, discolored, edematous. Orbital abscess.	Orbital abscess not infrequent—sometimes it is the occasion for case being brought to surgeon. Erysipelas appearing about the orbit. Ocular symtoms in influenza are suggestive. Glaucoma.
Reflex signs.			Infrequently retraction of muscles of back of neck, twitching of muscles of upper extremity. (Meningeal or cerebral irritation). Author's case.	Asthmatic attacks in rare cases.
Transillumination.	Illumination of pupil abolished except where excess of polypi has expelled pus. Opacity of cheek except as above. Absence of subjective light sensation in eye of affected side.	Results uncertain. Sinus sometimes very small or absent, which may give misleading dark shadow.		
Fever.	Usually moderate or absent.	Usually moderate unless acute retention of pus occurs.	Slight or none. When cerebral complications begin to supervene, temperature may range as high as 105 degrees F.	Usually moderate unless meningeal or orbital complications arise.

5—*A group passing through the cribriform plate* into the cranial cavity and anastomosing with the veins of the Pia Mater.

In addition to these five groups, *the plexus lacrymalis*, a most important set of vessels, penetrates the nasal and maxillary bones to empty into the nasal vein system on the face. This plexus establishes an additional, almost direct connection between the nasal cavities, the orbit and the structures of the face.

ETHMOID LABYRINTH.

Arterial Supply—*Ethmoidal arteries* anterior and posterior.
Posterior Nasal, a branch of the Nasal or Spheno-palatine from the Internal Maxillary.

Veins—*V. Ethmoidales Anteriores et Posteriores* pass upward toward the cranial cavity and the orbit, through the cribriform plate to the veins of the dura mater and the longitudinal sinus and to the veins of the orbital part of the frontal lobe of the brain, while others are deflected right and left through the anterior and posterior ethmoidal foramina to the ophthalmic vein which empties into the cavernous sinus.

FRONTAL SINUS.

Arterial Supply—*Ethmoidal Arteries* (anterior) from the Ophthalmic.
Branches from the *Spheno-palatine.*
Numerous minute *vessels from the bony walls* which help to bind the sinus wall and the lining membrane together.

Veins—Join the *V. Ethmoidales Anteriores et Posteriores.*

SPHENOID SINUS.

Arterial Supply—*Posterior Nasal*—branch of the Nasal or Spheno-Palatine from the Internal Maxillary.

Veins—Join the *V. Ethmoidales Ant. et Post.*

MAXILLARY ANTRUM.

Arterial Supply—*Alveolar* branch of the Internal Maxillary.
Infraorbital branch of Spheno-maxillary portion of Internal Maxillary.
Posterior Nasal, a branch of the Nasal or Spheno-palatine from the Internal Maxillary.

Veins—The major part of the blood is carried by a group of *nasal veins (Zuckerkandl's fourth group) which passes backward and upward* through the foramen spheno-palatinus into the canalis spheno-palatinus to join the maxillary veins on their way to the external jugular.

DISCUSSION.

Dr Wm. C. Posey, of Philadelphia, said that Dr. Holmes was to be congratulated upon adding another valuable paper to the literature of the ocular symptomatology of sinusitis. His earlier communications might properly be designated as pioneer explorations into what was then an entirely new field and Dr. Posey said we owed much of our present knowledge of the influence which sinusitis may have upon the eye to Dr. Holmes.

Dr. Posey went on to say that in Dr. Holmes' present communication he had done well to explain the vascular connections between the cavities and the orbit and his observations upon the inflammatory conditions and neoplasms of the cavities were of exceeding value by reason of his long and varied experience in this field of clinical work.

Dr. Posey stated that, as he had said elsewhere, the proper person to treat diseases of the sinuses was one who combined both rhinology and ophthalmology in his practice. He said that Dr. Holmes was such a one and he conceived that his grasp of the subject was therefore broader and more accurate than that of those who were strict rhinologists and strict ophthalmologists. But for various reasons, he said, the number of those who unite the two specialties in their practice was yearly becoming less and it was doubtful whether the association would be maintained many years longer; rhinologists relying upon their ophthalmological colleagues for the interpretation of ocular symptoms arising in the course of an inflammation in their territory, while ophthalmologists wait for the report of the rhinologist before arriving at a diagnosis in almost every case of orbital involvement.

He said that while this course might have its advantages, it also possessed many disadvantages, and a zone of neutral or disputed territory was left, in which the rhinological and ocular symptoms failed to dovetail into one another and the diagnosis of the conditions evoking the symptoms is obscured.

He thought there surely was need that ophthalmologists should better appreciate what was going on, and could go on, on the other side of the orbital walls, and that there might be almost as pressing a demand that rhinologists should be better acquainted with the relationship of the eye and its adnexa with the tissues which they treat.

Having this matter much at heart, and realizing the limitations of the knowledge of the average ophthalmologist of the sinuses, at the last meeting of the American Ophthalmological Society he ventured to insist that every annotator upon an orbit should be thoroughly familiar not only with the anatomy of the sinuses, but also with the various forms of operation which rhinologists have devised for their opening and treatment. He contended that this was necessary since he maintained that all external operations upon the sinuses should be performed by the opthalmologist. In order to explain his views fully, he thought it might not be amiss to quote a paragraph from the paper which he read at that time.

"In earlier years, all cases of sinusitis with orbital symptoms fell to the ophthalmologist. Rhinology had not been developed and practically nothing was known of the accessory cavities; but recently, with increased knowledge in diagnosis and treatment, rhinologists have absorbed the treatment of these cases, and among other procedures, have not hesitated to include the orbit in their field of operation, when they were unable to procure satisfactory drainage by intranasal methods. Incisions which lay bare the orbital contents, and dissections which often impair the safety of the eyeball and the optic nerve and seriously threaten vision, are practiced by them, and specialists in this branch who are informed but vaguely of the location of important ocular structures, proceed without hesitation in their operations in this alien territory. It is true, however, that the ophthalmologist is himself wholly responsible for this encroachment, for as a rule he has been entirely negligent in his appreciation of the fact that there are such things as sinuses, and that there are structures upon the other side of the walls of the orbit of which he should be better informed. For, beyond the recognition of the fact that nearly all abscesses of the orbit are secondary to sinus infection, until very recently, ophthalmologists were ignorant of many of the symptoms which sinusitis may evoke in their specialty. The writer would urge upon ophthalmologists, therefore, the desirability of a more intimate knowledge of the anatomy and

diseases of the sinuses. He does not intend by this that the rhinologist should be supplanted in the general treatment of sinusitis, but he is strongly of the opinion that where there are orbital complications or where external incisions must be practiced in the neighborhood of the orbit, the ophthalmologist should hold the knife, or at least superintend the work of the rhinologist. It goes without saying that the ophthalmologist, in his turn, should submit all cases in which he suspects sinus involvement, to rhinological examination, and in the event that the condition is reported amenable to *intra-nasal* treatment, that a transfer should be at once effected. Until the sinus is opened, it is impossible to determine the extent of cells involved, and in the event of extensive disease, the skill and resources of both ophthalmologist and rhinologist will often be taxed to bring the case to a successful issue. The services of both are essential if the patient is to receive the most skillful manipulation and consideration in diagnosis and treatment, and both should work hand in hand."

Dr. Posey went on to say that in a recent case of obscure ethmoiditis with orbital symptoms, Dr. George B. Wood and he, working literally hand-in-hand, brought an external operation to a successful completion, much more rapidly and satisfactorily, he thought, than if either of them had operated alone.

He said such associations of rhinologists and otologists were not always possible and it behooved each, therefore, to acquaint himself more thoroughly with the province of the other. Text-books on ophthalmology should contain a better description of the anatomy of the sinuses and a full account of the operations which are practiced upon them, while text-books on rhinology should describe at length the ocular symptoms which sinusitis may evoke.

He thought the rhinologist should be aware that a beginning sinusitis may occasion a stasis in the circulation of the optic nerve, which is readily discovered by the ophthalmoscope and by subjective tests; that paresis of one or more of the ocular muscles may result from an involvement of the nerve or nerves supplying them, as they pass along the outer wall of the sphenoid sinus, and that impairment of their function may result from direct involvement of the broad flat belly of the muscle as they lie in apposition with the walls of the sinus.

The significance of so-called prelacrymal tumors and abscesses should be appreciated and the swelling properly attributed to an in-

volvement of the lacryo-ethmoidal cells, and not to a distention of the sac.

He thought sinusitis was not an infrequent cause of asthenopia or symptoms of eye-strain. This may be occasioned by a change in the shape of the globe by direct pressure, either from inflammatory products in the orbit or by the bulging wall of a sinus. The asthenopia may also be originated as a consequence of faulty-acting extraocular muscles, or what is more often the cause, by an irregularity in the action of the ciliary muscle, either by reason of a vascular or lymphatic stasis, the result of a sinusitis, or by the action of toxines generated within the cavities upon the ganglia within the ciliary muscle. Blind spots and motes before the eyes are not infrequently caused by a derangement of the optic nerve, and troublesome and persistent conjunctival symptoms are often dependent wholly upon sinus disease.

He said it was most gratifying to those who were early interested in the importance of recognizing the ocular symptoms of sinusitis that this phase of the subject was finally beginning to receive the attention which it deserved. He stated, for example, that a recent number of the "Ophthalmoscope," a well-known British publication, was devoted entirely to the subject under discussion, while well-known German ophthalmologists who denied in communications to Onodi a few years ago that they had ever seen an inflammation of the optic nerve caused by sinusitis, now stated that inflammation of the cavities might occasion changes in the nerve, in terms as though the observation had arisen with them.

He was strongly of the opinion that as our knowledge of the pathology of the changes wrought in the nerve by sinusitis increases, we would find that a sphenoiditis or ethmoiditis was the basal element in not a few conditions in which the pathogenesis was obscure. It had occurred to him, for example, that an inflammation of the sheath of the nerve, resulting from a sinusitis, might result in a closure of the lymph space which surrounds the nerve, and so interfere with the exit of lymph from the eye that chronic glaucoma might arise. This was of course purely hypothetical, for so far as he knew no pathological study had ever been made of the sinuses or of the optic nerve adjoining the sinuses in any case of chronic glaucoma, but the association of chronic disease of the sinuses in several cases of glaucoma which he had observed had led

him to think that there might be a casual connection between the two.

In closing, he said, he should like to point out once more the value of an ophthalmological examination in all cases of sinusitis. In latent and obscure cases, the detection of a stasis in the circulation of an optic nerve will often be the first positive sign upon which we establish a diagnosis.

Finally, in the elaboration of the symptomatology of sinusitis and in the perfection of the treatment of diseases of the cavities, rhinologists and ophthalmologists, he thought, must work hand in hand, and ophthalmologists, especially, must make themselves more conversant with the anatomy of the sinuses and the various forms of surgical procedures which have been successfully practiced upon them.

Dr. Lewis A. Coffin, of New York City, emphasized the importance of urging upon pathologists that in all post-mortems the sinuses should be thoroughly examined. As to the frequency with which pneumonia, meningitis, etc., occur through the sinuses, more data were needed before this could be definitely determined. In the cases which he had observed of malignant and other tumors in the sinuses, the optic nerve was very much affected. He cited the case of a man sent to him on account of extensive dacryocystitis. Examination revealed osteochondroma involving both antra and completely filling the nasal fossae. Absolute atrophy of the nerve of the right eye was found, and the left eye showed all the premonitory symptoms of atrophy. Dr. Holmes' point about being careful in the region of the cribriform plate was well taken. If the condition demanded work in the immediate neighborhood of the cribriform plate, which was not so in ordinary ethmoiditis, a more open field of operation should be secured. Illustrative cases were cited. He heartily condemned the practice of cutting into tumors in these regions for the purpose of examination except immediately preceding radical procedure. It was unfortunate that most of these cases were hospital cases, and while they could be examined before operation, it was difficult to hold them afterward sufficiently long to know the end results of the eye condition.

Dr. Dunbar Roy, of Atlanta, Ga., said that his own practice, involving the diseases of both the nose and the eye, had afforded him a good opportunity to study their inter-relationship. That there is often a decided relationship between the diseases of the two or-

gans must be admitted, but that a universal relationship exists, or that most of the lesions of the eye can find their origin in disease of the nasal cavities or accessory sinuses, was, according to his own observations, a fallacy. There had never been a marked advance in any line of work where any particular operation or line of thought was constantly brought before the profession, but that in a reasonable time extremists appeared, and it was such men who brought advanced scientific work into disrepute. So it had been with the subject of the relationship of diseases of the eye and nose. Logical observers would, however, adopt such advancements as are valuable and discard such as are worthless. He considered the relationship in two divisions: (1) Diseases of the eye which are reflex neurologically from an abnormal or diseased condition of the nasal cavities; (2) Diseases which show objective changes in the eye, and which are dependent upon objective changes and direct extension of diseases in the nasal cavities. In the first category he placed neuralgic and asthenopic symptoms of the eye dependent upon such conditions in the nose as pressure from enlarged turbinates, spurs upon the septum, adventitious growths, and severe inflammations. While admitting that such conditions of the nose may produce neuralgia and asthenopic symptoms in the eye, he had not found them a frequent cause. That a small spur on the nasal septum which sometimes touches the opposing turbinate, especially in some congested states, should be placed in the category of a frequent cause of ophthalmic symptoms did not appeal to his reason or to his clinical experience. At the meeting of the Ophthalmological Society in 1900 Dr. Ewing and Dr. Sluder of St. Louis had read a joint paper on Nasal Conditions in Frontal Headaches. Dr. Ewing described as asthenopia eye symptoms characterized by increasing pain in the eyes after prolonged use, accompanied by tenderness at the nasal and under parts of the brow, and which could not be relieved by glasses when a decided error of refraction existed. Dr. Sluder had examined these cases and concluded that the pain was due to a congestion of the membrane of the nasofrontal and a pressure of the uncinate process against the bulla of the ethmoid, thus causing a closure of the infundibulum. This closure produced a change in the air pressure within the sinuses, and this inequality caused the symptoms of neuralgia and fatigue on using the eyes. Shrinkage of the membrane in milder cases, and resection of the middle turbinate in severer cases, was advocated for

the purpose of giving a free opening of the infundibulum. Since the presentation of this paper Dr. Roy had looked for similar cases, but with the exception of cases in which there was marked objective appearance of a polypoid condition of the middle turbinate and ethmoidal region, or marked hypertrophy with great pressure, he had been unable to find any such among the last 2,000 cases of refraction. He was inclined to believe the condition rare, at least in his section of the country. He had noted the symptom, to be sure, but always as a condition referable to the nose where distinct lesions could be found. It was his custom in all his eye cases to make a rhinoscopic examination to see if there were present other conditions besides errors of refraction. In such reflex neuroses he was convinced that a psychical element played a prominent part, and that the relief sometimes experienced by a cautery operation or the removal of a spur might be placed in the realm of suggestive therapeutics. However, real lesions of the nose and of the sinuses did occasion various subjective symptoms in the eyes. Frontal sinusitis, whether mucoid or muco-purulent, would occasion severe pains in the eyes. More painful to the eye, and especially on the movement of the eye, was an abscess in the sphenoidal and posterior ethmoidal sinuses following severe rhinitis. During the past winter he had treated ten such cases where the pain was so severe that anodynes had to be used for two or three days. All recovered under cocain and adrenalin shrinkage, together with warm salt water douches. Ziem had written a great deal upon this subject, attaching great importance to the reflex and direct action of diseases of the nose and accessory cavities to various conditions of the eye, and claiming that he had found diseases of the uveal tract, cyclitis, and even glaucoma to be dependent upon an accessory sinus infection. Ziem had claimed that in empyema of the maxillary sinus he had frequently found contraction of the field of vision. In severe cases such might occur, but Dr. Roy had been unable to confirm these observations. Kuhnt, Grunwald, Berger and others had reported reflex disturbances.

In the second category mentioned there was a direct extension of the pathologic process to the orbit or its contents from a diseased condition in the nose or accessory cavities. Four years ago he had had the pleasure of translating an article by Professor Oxenfeld, Professor of Ophthalmology in Freiburg in which he discussed orbital abscesses dependent upon their extension from and communica-

tion with the ethmoidal and frontal sinuses. Dr. Roy had reported two such cases at the meeting of the British Medical Association. In the first case the orbital abscess was taken for an orbital sarcoma because of the history and age of the patient and the existence of complete blindness. Operation revealed an abscess coming from the ethmoid cells. Vision was almost entirely restored. In the second case, a young man presented himself with proptosis and chemosis of the conjunctiva so marked that it protruded between the lids like a balloon. On examining the nose the middle turbinate on the affected side was found to be soft and pus was coming from the ethmoid cells. The turbinate was removed and the cells curetted and drained, with the result that in three weeks the eye was perfectly normal. When the thinness of the bone separating the orbit from the ethmoidal cells was considered it was surprising that the eye is not more frequently involved by direct extension. Nelson Black had reported a case of retrobulbar neuritis caused by a frontal sinusitis which entirely disappeared upon relief of the frontal sinus.

Professor Jansen said: In cases of malignant neoplasms I prefer the radical operation, where it is possible to remove all of the diseased tissue. I saw eighteen cases of neoplasms with disease of the antrum and ethmoids, and only three cases with disease of the frontal sinus. Of the former, I saw positive cure in four cases, including endothelioma, sarcoma, and carcinoma. There remain, unfortunately, a large number of inoperable cases. I will try Dr. Holmes' method in the future. It is astonishing how infrequently meningitis occurs, as a result of suppurative accessory sinus disease. In these cases there were severe headaches. Among the indications for operation, the fear of cerebral complications plays a very unimportant role. Furthermore, it is astonishing how seldom infection after a radical operation is to be looked upon as the cause of death, provided there has been no injury to the dura. I avoid exposing the dura, especially at great depths. Through the courtesy of Professor Hirschberg, I have the opportunity of seeing many eye diseases, complicated by suppurative accessory sinus affections. In several cases of amaurosis, the sight returned rapidly after intra- or extra-nasal operations. In some cases, however, I saw spontaneous improvement.

Dr. Thomas J. Harris, of New York City, emphasized the importance of the subject under consideration and called attention

to the lack of appreciation thereof on the part of rhinologists and ophthalmologists in general. A case which came under his observation at the Manhattan Eye and Ear Hospital was cited as an illustration of the dangers, so far as the eye is concerned, involved in the pathological conditions in the accessory cavities. The patient in question presented himself at the hospital two years ago, and a diagnosis was made of involvement of the accessory sinuses. Intranasal operations were performed, a large number of polypi removed from the interior ethmoidal cells. The patient left the hospital before the work was completed, however, and gave up treatment. He reappeared a year later in the eye department, with the report that while sitting at his table in good health, he had suddenly become blind in the left eye. The eye staff examined the case and found absolutely nothing through the ophthalmoscope. Two days later the case was referred to the rhinological department, where he was operated upon and extensive disease of the sphenoidal sinus was found. So far as the nose was concerned, the operation was successful, but so far as the eye was concerned, it was of no avail. Dr. Davis also, who examined the eye, could not determine what had happened. There was absolutely no fistula or breaking down of the wall of the orbit, and why the infection proceeded so rapidly was not known. The case emphasized the disregard on the part of ophthalmologists of the importance of these conditions, which should be referred at once to the rhinologist for operation.

Dr. William L. Ballenger, of Chicago, said that his experience did not agree with that of Dr. Roy, who had not found many refraction cases which were relieved by intranasal operation. Within the last two years he had had many cases referred to him by oculists, and he had found an undoubted relationship between the nasal conditions and the asthenopia present. After correction of the condition in the nose, whatever it happened to be, and carrying out the suction treatment according to Bier's principles, he had enabled the oculist to give perfect satisfaction with the use of glasses. A number of illustrative cases were cited, perhaps the most interesting of which was that of a man forty years of age who had double ethmoiditis and sphenoiditis. The ethmoidal cells were filled with polypi, and quite a large polypus was removed from the sphenoid. He operated upon the right side first, as this seemed to be objectively the worse. Vision rapidly increased, until the patient could count fingers at twenty feet. Five days later he operated upon

the other side, with the result that the patient became comatose, with involuntary action of the bowels. The light packing was removed from the nose, hot irrigation instituted, the patient rapidly regained consciousness, and is now at work, with a vision of 20-20 with glasses. In this case the vision was 20-20 with glasses before the operation, so that it might be said that it was just the same after operation as before the condition arose; but it must be borne in mind that this patient had been blind for only ten days before he was put under treatment, whereas in other cases cited blindness had existed for two months, so that the atrophy was greater. In one case, under observation of an oculist for two years previous to operation, the work on the nose did no good whatever. This emphasized Dr. Harris' contention that intranasal treatment should be instituted very early in the course of the disease.

Dr. Stephen H. Lutz, of Brooklyn, was convinced that there was a decided relationship between the nose and accessory cavities and the eye. He cited the one case of a woman twenty-eight years of age who had been blind since she was sixteen. She had consulted the leading ophthalmologists in this country and abroad, and had abandoned hope of recovering her vision. She had optic neuritis of the left eye. When she came under his observation three years ago, she had very noticeable ozena. He found a large hole in the sphenoid on the left side, and the probe revealed the absence of the external lateral wall and roof of the sphenoidal cavity. Here was undoubtedly to be found the explanation of the trouble with the eye.

Dr. John A. Thompson, of Cincinnati, reported two cases of paralysis of the third nerve following ethmoiditis, in each of which operation upon the ethmoid, and sphenoid in one case, resulted in perfect cure and complete restoration of the vision. For three years he had been watching a case with progressive suppuration, first in the ethmoid, then in the antrum, then in the frontal. The ethmoid was completely removed. With the oncoming of the symptoms in the frontal there was a sudden failure of accommodation in the eye on the affected right side. The severe headaches were not relieved by drainage of the frontal sinus until this failure of accommodation was recognized and a change made in the glasses. The glasses had to be doubled in refractive strength, first in the right and then in the left eye.

Dr. Joseph A. White, of Richmond, Va., referred to the connection between the nose and the eye through the lachrymal sac which, through its connection with the nasal duct, was really another accessory sinus. He had had a great many experiences in recent years which had led him to look upon the nose as a source of danger even when there was no trouble with the lachrymal sac. In two instances the loss of the eye had followed cataract extraction despite every possible precaution. In each case subsequent examination showed the nose to be filled with pneumococci. With no apparent trouble in the nose, no secretion, no disease of the lachrymal sac, the eye was lost because of infection which evidently traveled up through the nasal duct. He had been amazed to find in how many cases the conjunctival secretion is infected where there is no apparent disease of the nose.

He advocated preliminary bacteriological examination of the conjunctival and nasal secretions before operations necessitating opening the eye-ball, and the obliteration of the lachrymal sac, if virulent germs were found in the secretions.

Dr. Joseph Beck, of Chicago, called attention to the fact that post-mortem examinations of the accessory sinuses are usually made by general pathologists who have no special knowledge concerning this region, and who consequently are unable to do satisfactory work. It had been established by Wingrave and others that the finding of post-mortem fluid was not necessarily an evidence of disease of the accessory sinuses, but a post-mortem change. He had been making his own post-mortem examinations of the sinuses and had found it far more satisfactory.

Referring to the question raised by Dr. Holmes as to why infection from the nose to the eye is so rare, he called attention to the explanation of Dr. Dail, of Prague, who said that the angularity of the ophthalmic vein governs the traveling of infection. This had probably never been proven, but when the suggestion was made at the Russian Congress, it was very warmly received.

Dr. Posey called attention to the fact that headache and many ophthalmic symptoms were not infrequently found to be due to sinusitis, even when there were no nasal signs of the latter condition, and in the operative treatment of such cases both the rhinologist and the opthalmologist should be present, unless the surgeon had had experience in the surgery of the eye, nose and throat.

PAPER:

RHABDOMYOMA OF THE NOSE.

DERRICK T. VAIL, M. D.

Rhabdomyoma (Myoma Striocellulare, Rhabdomyoma Sarcomatodes, or Rhabdosarcoma) has been observed in the various organs of the body, most frequently in the kidney, uterus, rectum, stomach, testicle, once in the parotid gland and once on the root of the nose between the eyebrows. A single case like the following becomes interesting only on account of its extreme rarity, and, believing that all unique cases should go on record, I beg to report it. So far as I can ascertain it is the first case of its kind on record.

The patient, Mrs. L., first consulted me regarding the present trouble May 7th, 1906. She gave the following history:

Six weeks ago had the "grip," affecting the respiration apparatus and accompanied by considerable stoppage of the nose with twinges of pain in the ears. Was treated by her family physician, who, meeting with little success in relieving her of her nasal symptoms and recognizing that a change of climate would be beneficial in a general way at least, advised her to go to Atlantic City. She followed his advice, staying two or three weeks, and returned feeling much better as regards her general condition, but still complaining of nasal stenosis on the left side. She called to see me regarding the symptom, which was not painful, but annoying.

Status Praecens: Patient a spare but well nourished lady of fifty-eight years of age, presenting no external evidences of ill health and giving no symptoms other than complete stoppage in the left nares.

Examination: Right naris normal; left revealed no breathing space whatever, the turbinal body being in tight contact with the septum, and the attempt to introduce pledgets of cotton saturated with cocaine and adrenalin solution met with failure. The

stenosis began just within the vestibule of the nose and seemed to be entirely due to a swollen inferior turbinate. The middle fossa could not be seen. Post-rhinoscopic examination revealed the choana of the left side to be completely filled with a mass of pale tissue, light gray in color, which felt like a nodulated mass of tough tallow. The transillumination test was negative, the hearing unimpaired and the eye normal in all respects. My associate, Dr. Lamb, examined the case with me and we agreed that it "looked suspiciously like a sarcoma," although it lacked the reddish color of the sarcomata and did not bleed when wounded. We advised removal of the growth and gave a guarded prognosis.

The Operation: On June 4th, 1906, I attempted to cocainize the left naris and made more or less futile attempts at getting solution of adrenalin within the nasal cavity, but very little, if any, could be crowded along the septum on account of the impacted mass of tissue. Scissors could not be passed without puncturing and tearing the specimen. A fine straight nasal saw was passed under the growth along the floor of the nose and an attempt was made to engage the inferior turbinate in the cutting stroke. Meeting with no resistance that felt like anything, the saw was withdrawn. A nasal snare loaded with a large loop of No. 4 piano wire was insinuated along the septum until it passed the growth and the wireloop could be felt in the pharyngeal vault with the finger. The tip of the index finger of the left hand was passed through the loop of wire in the pharyngeal vault and made to pass up over the summit of the growth, and then by drawing the wire home, it slid along the finger and thus entire tumor mass was engaged. I expected to meet with considerable resistance by this large mass of tissue engaged in the wire-loop, but on drawing the wire it was delivered in the canula of the instrument without any apparent resistance. On attempting to extract the mass, I encountered considerable difficulty. I could not extract it forwards; it was too large and any attempt to grasp the front end of the mass met with utter failure for the reason that the tissue was so friable that it could not be grasped. I succeeded finally in getting it out piecemeal by the various means at my command, and extracted a wineglassful of fragmentary masses of the tumor. There was no bleeding and inspection seemed to show a thorough removal.

The specimen was hardened and sectioned by Dr. Lamb and was thought to be a large spindle-cell sarcoma by those who examined it. This information was given to her brother, who desired the specimen sent to any expert pathologist I might choose with a view of getting a positive diagnosis. Two pieces of the growth were sent to Dr. Wm. H. Welch, of Johns Hopkins University, Baltimore, and I beg to read his report in full.

Examination of Tumor from Nasal Cavity, for Dr. D. T. Vail.

The specimen is a solid grayish white elongated mass of tissue, about 1½ ctm. long and 5-8mm. broad, and of irregular shape. A part of the free surface is smooth as if covered with mucous membrane. The cut surface appears smooth and uniform. The tissue is preserved in formalin. There are two parts of the growth in the bottle, from each of which sections were cut. *Microscopical sections* show over a small area remnants of the epithelial covering in the shape of cubical cells, but over most of the surface the epithelial cells are absent. The rest of the section is composed entirely of neoplasm.

The tumor consists essentially of interlacing bundles of fibers and cells, which appear cut in different directions. The characteristic and predominant cells are elongated, band-like and fusiform cells, but large, round, oval, irregular and branching cells also occur. There are also cells with large, irregular, budding nuclei and with multiple nuclei. All of these cells probably belong to the same group and are embryonic, striated-muscle cells in various stages of differentiation or entirely undifferentiated.

The most highly differentiated cells are long, ribbon-shaped cells with one, rarely two or more, vesicular nuclei and with bodies which stain deeply with eosin. These bodies present often distinct longitudinal, delicate fibrillation, and less frequently, but in some cases distinctly, cross striation, especially at the margins of the cells. The ends of the cells appear continuous with delicate fibrils which can be traced into the surrounding tissue, either as separate fibrils or as a network of anastomosing or interlacing fibrils. These cells, which can be positively recognized as embryonic striped muscle, occur in moderate numbers scattered throughout the growth, but are most numerous in a small area near the margin of the section.

By the identification of the foregoing cells as muscular, the interpretation of other cells of the tumor as less differentiated

muscular cells becomes easier. Thus there are long, flat cells with longitudinal but without transverse striation, these cells being much more numerous than those with cross striation; similar cells with smooth or granular undifferentiated cytoplasm, which, as in the forgoing cells, stain deeply with eosin; large and small, round or irregular cells with similar cytoplasm, and branching or even spider cells with delicate processes continuous with a reticulated stroma. The margins of many of the cells described appear as a glistening, single or double contoured, refractive line.

These various cells are arranged for the most part parallel to each other in interlacing bundles. When such a bundle is cut across, it sometimes happens that the contents of the cells drop out, leaving the refractive borders, so that a peculiar reticulated or honeycombed structure results, or it is possible that this appearance may be due to dissolved glycogen. There appears, however, also to be in some places a genuinely reticulated stroma, and it can sometimes be seen that fibrillar processes from the cells are continuous with this reticulum. Exceptionally on cross sections of the muscle-cells, Cohnheim's fields can be made out.

There are many elongated, round or branching cells which are without any suggestion of a muscular origin. Whether these should be interpreted as connective-tissue cells or entirely undifferentiated embryonic muscle-cells, is a matter of arbitrary judgment, but I am inclined to the latter interpretation, as all transitions between these and recognizable muscle-cells exist.

The giant cells are doubtless of muscular origin, although this too cannot always be demonstrated.

The tumor as a whole is fairly rich in cells, although in places the large amount of cytoplasm and fibrils makes the nuclei appear far apart. There are a few irregular areas of coagulative necrosis. Larger blood-vessels are not seen, but there are several wide lymphatic vessels. Genuine connective tissue is not abundant.

Diagnosis: Rhabdomyoma Sarcomatodes.

Remarks: Rhabdomyomata as rich as the present tumor in undifferentiated cells, resembling sarcoma cells, are usually designated Rhabdomyomosarcomata, but I prefer the above designation, as it seems to me probable that these undifferentiated cells are of muscular rather than connective-tissue "Anlage."

These tumors undoubtedly spring from misplaced, embryonic germs of striped muscle and are often developed at birth or in

early life, although they may appear later. They are most common in the kidney, next in other parts of the genito-urinary tract, but they have been observed in various other parts of the body. In Benenati's collection from the literature of sixty-four cases (Virchow's Archiv. 1903, Bd. 171, p. 418) there is none within the nasal cavity, and I am not aware of any instance of this localization previously observed.

The majority of reported rhabdomyomata have not been malignant in the sense of metastasizing or of invasive, infiltrating type of growth. They may, however, be malignant and metastasize, especially when the growth is rich in sarcoma-like cells. I have examined specimens from two such cases of malignant rhabdomyoma, one being of the vagina and the other of the bladder.

WILLIAM H. WELCH.

There was no history of syphilis, but the patient was put on K. I. and Hg. and remedies were pushed to the point of saturation without any result.

In the meantime, the nasal cavity began to fill again and the transillumination test showed left side of the face dark. Another operation similar to the first was advised, as it was seen that the growth involved the upper turbinal wall. This was consented to and on October 15th the lateral wall of the inferior and middle meatus of the nose was rid of the same kind of growth as was found at the first sitting, and the middle turbinal body was entirely removed. It was observed at the time of the first operation that the lower turbinated bone was entirely absent —digested—absorbed—transformed or rarefied; at any rate, there was no bony framework of the inferior turbinated present. At the second operation the conformation of the middle turbinated was plainly made out, but on looking at the specimen, you will see that it, too, was rapidly losing its bony framework, being transformed into this tallow-like mass of nodulated tissue, which you can tear apart in shreds like a mass of soft papier-mache. The cavity was as thoroughly cleaned out at this second operation as possible. There was no bleeding at any time, either at the time of operation or afterward.

Soon after there was evidence of recurrence and I called a consultation with Dr. C. R. Holmes, who agreed that the growth was of unusual character, was probably filling the antrum and that a radical operation should be performed. There was no

facial deformity and no subjective symptoms aside from complete blocking of the nose on that side. As the patient naturally dreaded the heroic operation proposed, it was decided to send her to New York for further advice, and Dr. Emil Mayer and Dr. R. C. Myles were consulted. I will submit extracts from their letters.

Extract from Dr. Mayer's letter:

Mrs. L. called to see me on February 1st, and I have since seen her daily for three days for the purpose of making a full report to you.

With your history to go by, the growth in her left naris, which practically occludes that side, is readily recognized as a neoplasm. It does not shrink under cocaine, nor adrenalin, is hard and bleeds readily. Nevertheless it seems to be made out to be limited to the site of the turbinate only. While it may be encroaching on the ethmoid cells and antrum, examination fails to show that. The transillumination test carefully made shows no difference on either side.

With the positive diagnosis of neoplasm, it seemed best to me to see if there was any change from the conditions found by Dr. Welch last June, and consequently I removed a small piece of the growth and referred it to the pathologist at Mt. Sinai Hospital. I enclose his report, and you will see he agrees with Dr. Welch in his report, although he did not see the striae that made it a rhabdomyoma. There are good reasons for this. You sent a large specimen, I sent a small one; yours was of the original growth, while mine was of the recurrence.

The main point is to decide if in the past six months there have been any changes in the growth indicating greater malignancy. This may now be definitely negatived.

The diagnosis is a sarcoma of rather benign tendency and we have therefore every reason to believe that its thorough removal will effect a cure.

I have given careful thought to your query as to treatment. Except for a history of several miscarriages there is no reason to suspect syphilis, and I believe you have given her full doses of KI with negative results; so I eliminate that. I have considered the possible value of X-rays and I greatly doubt their efficiency.

It seems to me therefore after careful examination, that her best interests will be in an operation which will remove the

growth and the adjacent affected parts, not necessarily all of the half of the maxillary bone.

Mrs. L. complained of pain or pressure over chest and radiating into the arm. I found no chest abnormality and no glandular enlargement.

I have therefore only to verify in every way what you have done, to agree with you in the treatment heretofore, and finally to thank you for the opportunity to see so interesting and unusual a case. Very sincerely yours, EMIL MAYER.

The pathologist referred to in Dr. Mayer's letter was F. S. Mandlebaum. He made the following report:

The tumor from the inferior turbinate sent for examination is a small, hard, oval-shaped nodule, measuring from 6-8 mm. in diameter, without any special distinguishing features on the surface. On cutting it in half, its surface is greyish in color and quite homogeneous in structure.

Microscopic Examination: When viewed with the low power, the entire central portion appears more or less degenerated and stains quite poorly as compared with the peripheral parts. With the higher power the central portion shows that the degenerative process has ceased before complete necrosis, and the component structures are still faintly visible The peripheral zone shows distinctly the character of the growth, which extends quite to the surface and is not covered by mucosa.

The growth is composed almost entirely of well-defined cells with more or less parallel margins and of variable lengths. The ends of these cells are frequently drawn out to a thin filament, and often merge into the conective tissue stroma. These cells stain readily with eosin and with picric acid. No striations can be seen. The nuclei of the cells are vesicular and very rich in chromatin, and vary greatly in size and shape, but for the most part are rather broad and oval and resemble those of the heart muscle. Here and there scattered between these cells are other large cells of irregular shape, with large nuclei, and occasionally a giant cell with multiple nuclei is found. The cells that comprise the greater part of the tumor are unquestionably muscle cells in various stages of development. Where bundles are cut transversely, the typical appearance of muscle tissue is apparent. In those parts where the muscle fibers are less numerous, a stroma of delicate connective tissue fibers, branching and inter-

lacing, is seen. In the meshes of this stroma a few mononuclear leucocytes are present. No distinctly embryonal connective tissue fibers can be made out. No mitosis, either typical or atypical, is present. The central portion of the tumor, as far as it is possible to make out, consisted of similar structures prior to its degeneration.

Blood vessels are not numerous in the section. Those that are seen are thin-walled and show no lesions.

Diagnosis: Myosarcoma (leiomyosarcoma).

With many thanks for your courtesy, I am

Very sincerely yours, F. S. MANDLEBAUM.

Dr. Myles' report on the case was as follows:

In my opinion Mrs. L. has a malignant growth in the nose. The anterior area could not be properly observed on account of the closeness of the septum to the outer wall and the considerably bruised edema probably due to manipulation. The inferior turbinal can be easily observed through the posterior nares. The posterior half of the inferior turbinal is larger than it should be; its middle and anterior part appears nodular and roughened (by posterior rhinoscopy aided by retraction of the soft palate.) The neoplasm is extending outward, upward and forward toward the nasal process of the superior maxillary bone and the nasal vestibule.

I would advise a radical operation by making an incision beneath the eye down to or through the lip and resection of a part of the superior maxilla. The growth seems to be confined to the anterior half of the nasal fossa. Of course the tissues should be removed a certain distance beyond the apparently healthy lines.

Very sincerely yours, ROBERT C. MYLES.

It was decided to have Dr. Jos. Ransohoff, a general surgeon of Cincinnati, perform the operation, as it was thought the growth may have extended to such territory that the upper jaw might have to be removed together with the entire accessory sinus framework on that side. On February 9th, Dr. Ransohoff examined the case thoroughly and agreed that the external operation (radical removal) was necessary.

On February 12th at the Jewish Hospital the patient was operated on as follows:

Anesthesia by Dr. Iglauer—chloroform. Preliminary laryngotomy and tube inserted; pharynx stuffed with gauze. This was

decided expedient on account of the likelihood of dangerous and annoying hemorrhage and to facilitate the giving of the anesthetic. An incision was carried from the inner angle of the left eye down the side of the nose around the ala nasi and thence outward parallel with the lip. The periosteum was stripped back and the front wall of the antrum together with a part of the nasal bones were removed with the bone forceps. It was then seen that the tumor mass completely filled the antrum of Highmore and was not adherent at its limitations.

The removal of the entire mass was easily accomplished by simply lifting it out of its nest. The confining walls were thoroughly inspected for ingrowths of the tumor, but none were found. The cavity was packed with iodoform gauze and the wound closed by interrupted sutures. The tracheotomy tube was removed and sutures applied, closing the opening in the larynx.

During the entire operation, which occupied only about twenty five minutes, the patient had a bluish, cyanotic color, her pulse was thready and the heart lacked tone. The patient rallied from the anesthetic so far as the regaining of consciousness was concerned and did not complain of her respiration. Her breathing was deep and normal, but the heart did not rally and in spite of the employment of every known means of restoring the cardiac power, the pulse continued small and thready, and sixty-eight hours after the operation the patient died.

Stengel, in his 1906 book on Pathology, speaks of Rhabdomyoma as "containing more or less striped muscle fibers, usually a small amount, the main body of the tumor being made up of sarcomatous cells.

Etiology: It is thought to be a congenital defective development, and usually occurs in early life and in situations where unstriped muscle fibers do not normally exist. The kidney is the most frequent seat of involvement.

Structure: The microscopic appearance is usually that of a spindle-cell sarcoma, containing more or less striped muscle fibers. There are elongated spindle-shaped cells, partly striated and suggesting embryonal muscle tissue and rarely more fully developed muscle fibers. Large areas may contain no muscle fibers at all, while certain portions are richly supplied.

The tumors are malignant in proportion as the sarcoma elements are predominant, but metastases are infrequent.

I have no doubt that rhabdomyoma of the turbinates has been encountered, but has never before been recognized. The appearance as it exists in the nose is that of a lardaceous or suet-like nodular mass of friable nonvascular tissue. These tumors going to the laboratory for microscopic diagnosis are called large spindle-cell sarcomata.

When one considers that within the nasal structures striped or voluntary muscle fibers do not normally exist, he wonders that such a tumor in this locality could occur. The only plausible theory for its occurrence in a locality of this sort is that one of the neighboring voluntary muscles, such as the levator labii superioris alaquae nasi or depressor anguli nasi, has a prototype embryologic, misplacement lying dormant within the nasal cavity and for some reason took on active sarcomatous growth late in life.

A complete bibliography on the subject is published in Ziegler's "General Pathology," 1903, page 406.

DISCUSSION.

Dr. John J. Kyle, of Indianapolis, Ind., said the discussion of a tumor of this character involved a discussion of tumors in general in this region. One of the etiological factors in the production of tumors of the nasal and accessory cavities was probably irritation, most commonly produced by acute catarrhal or acute purulent inflammation. He had seen three cases of malignant disease of the maxillary antrum. Particular attention should be given to the sinuses because it was from this source that irritation was derived. Dr. Vail, in speaking of the treatment of his case, reported having performed preliminary tracheotomy; in Dr. Kyle's cases, where half of the superior maxillary bone was removed, it was not found necessary to resort to tracheotomy. Tracheotomy may add to the danger of the operation and be of no particular value.

Dr. Christian R. Holmes, of Cincinnati, who had seen the case in consultation, agreed with Dr. Vail that removal of the tumor was the only proper course to be followed. The operation performed was in every way perfect, but the patient was in a desperate condition. He had never seen a similar case. Whenever possible, he would avoid making a tracheal incision in such cases. With the Young inhaler, or the modified inhaler of Gwathmey, by plugging the posterior nares most of these cases could be operated upon successfully without tracheotomy.

PAPER:

FURTHER OBSERVATIONS ON THE ROENTGEN-RAY EXAMINATION OF THE ACCESSORY NASAL SINUSES.

BY E. W. CALDWELL, M. D., NEW YORK.

The members of this society are all familiar with the results obtained by examination of the accessory sinuses by means of the Roentgen-ray, and you are all better able than I am to estimate the practical value of such examinations. I shall, therefore, have little to say of the clinical aspect of the subject, but shall consider in this paper chiefly the Roentgen-ray technique.

In the beginning it may be well to consider for a moment some of the very prevalent erroneous ideas concerning X-ray examinations in general. The impression still prevails that the X-ray is a very powerful or special form of light by means of which we may photograph bones and other anatomical structures ordinarily hidden from our view. The wonderful development in photographic apparatus and materials, and the well merited popularity of amateur photography, have made it easy for us to suppose that X-ray work is merely another species of photography. Enterprising manufacturers of X-ray apparatus have found it to their interest to encourage the belief that possession of their wonderful appliances would bring within the reach of anyone the pleasant pastime of making the so-called X-ray photographs.

This misconception of the principles involved in X-ray diagnosis is, in a great measure, responsible for the disappointments of those who have attempted to use the X ray without knowing how or when it may be of service to them.

In the first place the X-ray does not give us a photograph or even a picture that is like any other picture we know. We are so accustomed to photographs that we do not stop to analyze the physiological processes involved in interpreting them. We do not

realize that the ability to obtain accurate impressions of the subject represented by a photograph has been acquired by years of experience. Travelers tell us that uncivilized people acquire very slowly the ability to interpret an ordinary photograph, and that it may take months for a Kaffir native to learn to recognize a portrait of one of his companions.

In viewing a photograph or a picture, the retina receives an impression of light and shadow, and perhaps of color, which is a more or less perfect imitation of the effect which would be produced by viewing directly the object portrayed.

The X-ray gives us projections which sometimes strongly resemble pictures, but these projections never portray an object as the eye would see it. It is therefore unsafe for anyone to attempt to interpret such a projection as he would a picture. We must guard against false impressions of perspective, and must adopt a new view point, which for many seems to be as difficult to acquire as for the Kaffir native to grasp the meaning of an ordinary photograph. When one has acquired this point of view the lights and shadows of a skiagraph represent only results from which he must deduce the cause; he knows that they do not present a front view or a back view or any other *view*, and the expressions "X-ray picture" and "X-ray photograph" cause him weariness and depression. When this point of view has become more prevalent the pictorially excellent skiagraph of a hand or a foot made by some enthusiastic amateur will no longer excite wonder, and the photographers, electricians and janitors who now make the so-called X-ray photographs in many hospitals, will have their activities transferred to other fields where they will be less a menace to the public health.

X-ray appliances are still crude and comparatively undeveloped, and they have not approached the degree of perfection of the apparatus that has brought photography within reach of the amateur. In recent years the demand for more perfect results and the enlarging field of usefulness of Roentgen-ray diagnosis has made it necessary to use more and more elaborate and expensive apparatus. In 1897, when the art was new, one could have bought a fair X-ray outfit for $200, and the best tubes then available did not cost more than $15. These outfits were more or less useful toys, and the same criticism applies to many of the outfits that are now sold to physicians who are inexperienced in this work. With the best

appliances now obtainable difficult skiagraphic work, such as we have under consideration, requires a degree of skill and judgment comparable with that which is expected in any other medical or surgical procedure, and greater than we can reasonably expect from the hospital photographer or janitor.

In the less difficult applications of the X-ray a very poor skiagraph may give useful information, and may, therefore, be mistaken for good work. A difficult and brilliant X-ray diagnosis is often less appreciated than a pictorially good plate of some easy subject like a shell fish or a mummy's hand.

An important feature of the diagnostic use of X-rays in the nasal tract is that in this field mediocre work is useless or worse, and the very best that can be done often leaves much to be desired. Many operators who have believed themselves expert because they could make useful skiagraphs of fractures, foreign bodies, teeth, etc., have had so many failures in frontal sinus examinations that their work in this field is of no value.

There is no mystery about this work, and no justification for the statements occasionally heard to the effect that success in this field depends upon some trick which is the secret of a few. It is true that the majority of owners of X-ray outfits are unable to make good skiagraphs of the frontal sinus region, but it is also true that such work is done satisfactorily by practically all of the really competent Roentgen-ray specialists, although probably no two of them employ the same tricks of technique. Excellent work has been done also by a few surgeons and rhinologists who have given the subject the serious attention that it demands and deserves. The comparatively few successes and many failures in the work cannot be charged to secrets of technique, but result partly from the fact that the amount of attention necessary for good work has generally been underestimated.

The difficulty of the work is easy to understand when we remember that the skiagraph is a composite shadow of everything lying between the source of rays and the photographic plate, and that the different objects cast stronger or weaker shadows, according to their relative thickness, their density, and the atomic weights of the elements of which they are composed. A skiagraph of a hand is essentially an X-ray shadow of the bones, which compose a large part of it, and which are but thinly covered by soft tissues, much less resistant to the rays. Such a skiagraph may be easily made

with almost any X-ray outfit, and it matters little whether the rays used are of high or low penetration, because the contrast in X-ray opacity of these tissues is great. In making a skiagraph of the frontal sinus region the rays must pass through the head in a long diameter; they must traverse the occipital bone and the brain, but the shadows of these thick and dense structures must not obscure the shadows of the relatively thin bones forming the sinus, or those of the fluids contained therein. It will be evident, therefore, that for this work not only must the penetrating quality of the rays be regulated accurately in order to secure differentiation, but that in order to produce sufficient effect upon the photographic plate, the rays must be powerful, and the exposure long. To obtain all these conditions requires careful manipulation and the best appliances obtainable. The chief difficulty is to obtain tubes that may be operated for these long exposures without breaking, and without too much change in the quality of the rays emitted. No two X-ray tubes are exactly alike, and in practice I find that only about one in five or six of the tubes I buy is suitable for frontal sinus examinations, although for most work they would all be useful.

With a good tube and exciting apparatus a skiagraph of a wrist or hand may be made with an exposure of one second or less, but under the same conditions a frontal sinus exposure will require from 20 to 50 seconds. During such short exposures as one to five seconds, the ordinary tube will not get hot enough to endanger its life or seriously interfere with the quality of the rays emitted, but only a very good tube will deliver continuously the same quality of rays during the long exposure necessary for a good frontal sinus plate. After such an exposure it is advisable to give the tube a rest of at least half an hour to let it cool off before using it again. When the tube gets hot, gases are driven from the glass and metal surfaces, reducing the vacuum, and with it the penetration of the rays. For this reason I prefer large tubes with eight-inch bulbs, and very heavy targets. A given amount of gas liberated in a large chamber of course produces less change of pressure than in a small one, and the rise of temperature in a large heavy target is less than in a smaller one.

The resistance of the tube must be adjusted for rays of high enough penetration to pass through the skull and brain, but rays of too high penetration will not give the contrasts that are necessary. I think that the best penetration is about nine or ten on the

Benoist Scale. With this degree of penetration there is danger of cracking or puncturing the tube during a long exposure, and I have frequently ruined, in a single sinus examination, a tube that might have been used safely for a hundred or more skiagraphs requiring less prolonged exposure. It seems to me that in every successful sinus examination the tube must be strained much beyond the limit of safety. On account of this strain the exposure should be shortened as much as possible by using the fastest plates obtainable. My best work has been done with plates made in Germany, but for the last year these plates have proven so unreliable that I have had to abandon them altogether and use American plates, which are fully 25 per cent slower.

The development of the sinus plates does not differ from that of any other skiagraph. Developers giving density are to be preferred, but no two operators agree as to just what is the best developer. My best results have been obtained with pyrogallic acid; next to this I prefer hydroquinone.

When one is provided with the best X-ray outfit obtainable, and has learned to operate the tubes not merely to give X-rays, but to deliver rays of approximately the right degree of penetration for a sufficiently long period, he may properly consider the details of arrangement of plate, tube and subject necessary to show the nasal sinuses to best advantage. My own views on the subject were presented in a paper read before the American Roentgen Ray Society in September, 1906, and published in the American Quarterly of Roentgenology for January, 1907. Since that time I have not modified my technique, except to lengthen the exposures, and thus compensate for the slower plates. Not only has this increased the wear and tear on tubes, but it seems to me that the results I get now are not so good as with the fast plates.

It is, of course, necessary to make a transverse projection in order to show the depth of the frontal sinus. In this projection the rays pass through a short diameter of the skull. The same degree of penetration (Benoist 9 to 10) as for the antero-posterior projection will give good results, but for this plate the quality of the rays is comparatively unimportant. The exposure need not be more than half so long as for the antero-posterior projection, and there is therefore much less strain on the tube. The plate may be supported in contact with the side of the head and in a plane parallel to the median plane

of the skull. The ray which is perpendicular to the plate should pass through the glabella. This projection is comparatively easy to make, and there should be few failures and little breakage of tubes. The knowledge of the antero-posterior depth of the frontal sinus, obtained from the transverse projection, is often useful in arriving at a correct interpretation of the antero-posterior projection. If the sinuses are deep the quantity of fluid they may contain is greater and the shadows cast by the fluid will be correspondingly stronger. When the sinuses are very shallow it will sometimes be impossible to decide whether they contain fluid. It has been proven that an edematous lining membrane will cast as dense a shadow as a collection of pus or other fluid containing the same amount of water, and this should always be borne in mind.

The antero-posterior projection is the one which shows the size and outline of the sinuses and the number and position of their septa, and enables us to ascertain the presence or absence of fluid, either free in the cavity or in an edematous lining membrane. This projection requires the long exposure, and is the one in which the operator is most likely to ruin his best tube, and to lose that cheerful but dignified composure that should always characterize the medical man.

In making this exposure it is important to use rays of as nearly as possible the degree of penetration before mentioned and to maintain them for from 20 to 50 seconds, according to the thickness of the skull and the power of the exciting apparatus. The plate is placed in contact with the face of the subject, and the head as well as the plate and tube must be supported so that there will be no movement during the exposure. With the average skull the frontal sinus will be best shown when the ray passing through the glabella in the median plane makes an angle of about 25° to 28° with the plane of the base of the skull. In making this exposure it matters little whether the patient lie with face resting upon the plate with the tube above and behind, or on a canvas stretcher with tube underneath and plate supported above and in contact with the face. Personally I consider the last mentioned position more convenient, but it requires special appliances which are not obtainable in the instrument shops.

In most cases the proper direction of rays may be obtained with sufficient accuracy by placing the tube so that the glabella, the parietal eminences and the target of the tube are approximately

PLATE I.

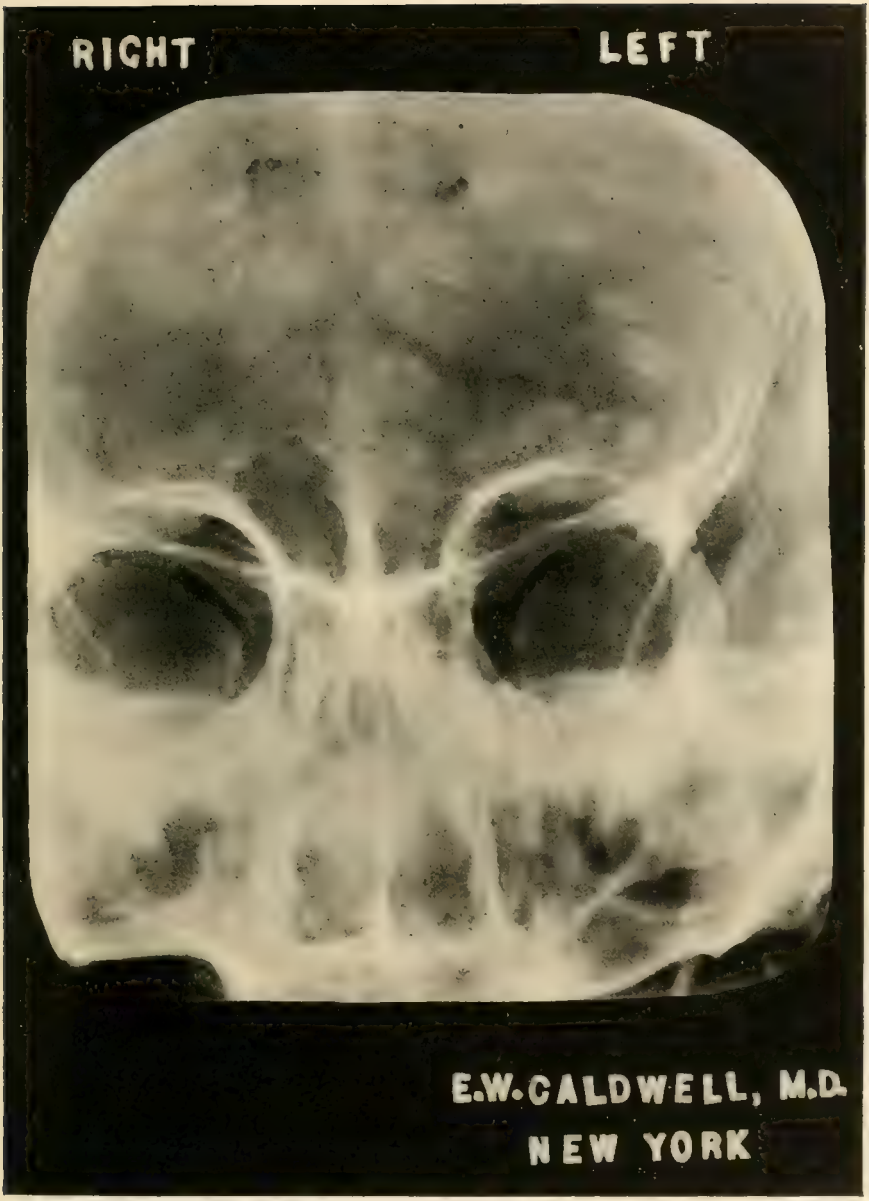


PLATE II.

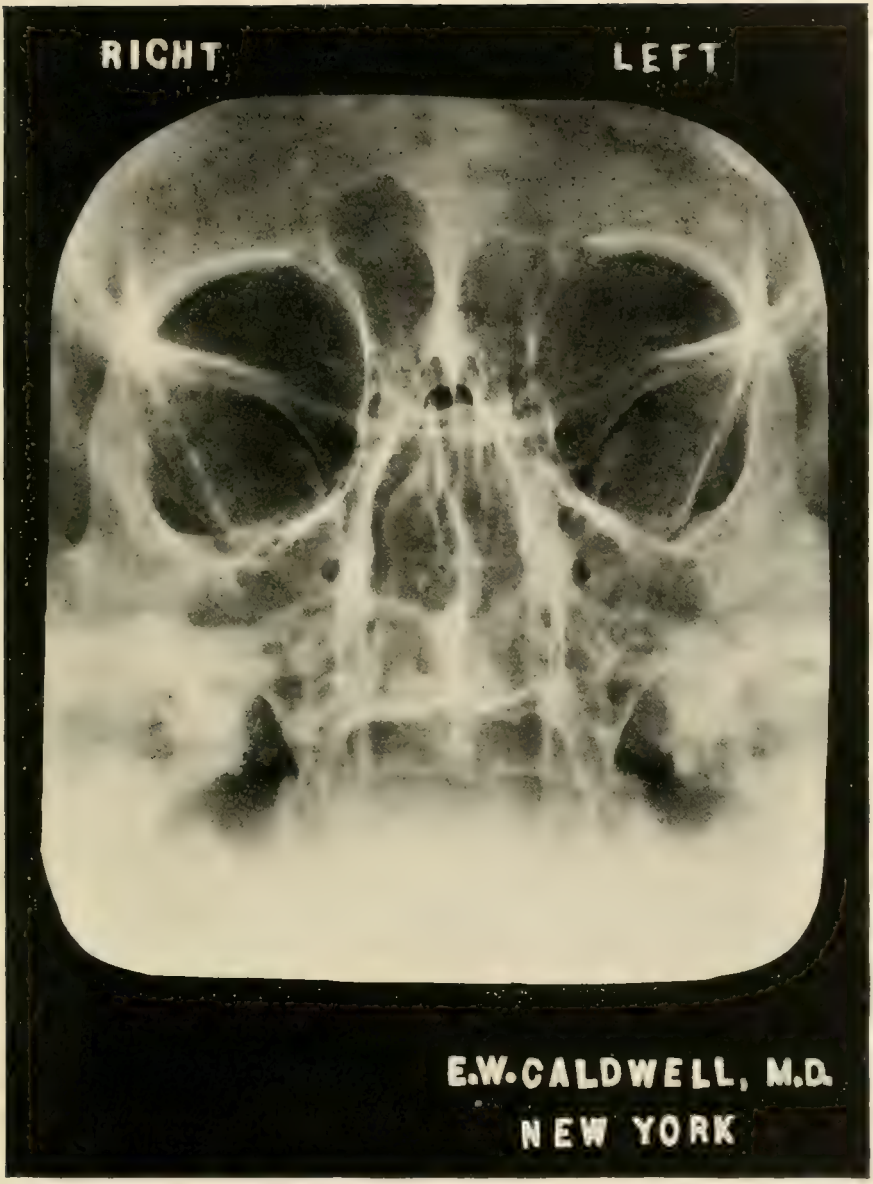


PLATE III.

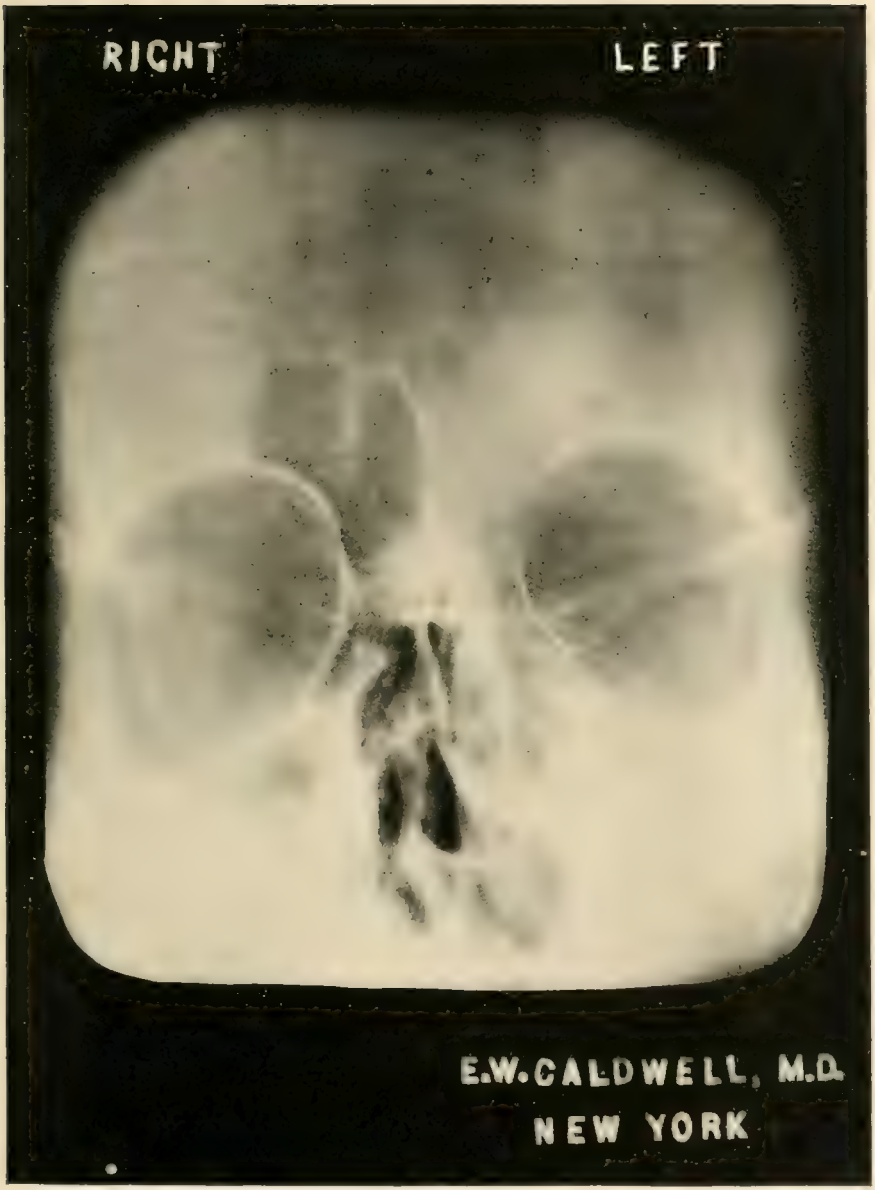


PLATE IV.

PLATE V.

in the same plane, the source of rays, of course, lying in the mesial plane of the skull. Failures seldom result from inaccuracy of this angle, but most of them are due to inability to obtain from the Crookes tube the right quality of rays for a sufficiently long period.

In conclusion I have to say that perhaps this paper has unduly emphasized the difficulty of the work. Some operators have apparently had less trouble with it than I have had. From my own experience, however, I feel fully warranted in recommending that these examinations be made by Roentgen-ray specialists when such men are available. Many rhinologists who do not live in large cities find this impossible, and may employ this method of diagnosis only by equipping their own offices with X-ray outfits and doing the work themselves. The writer hopes that they may derive some help from the suggestions in this necessarily incomplete paper. It is to be hoped and expected that future developments in appliances and technique will remove many of the difficulties that have harassed the pioneers in this field.

EXPLANATION OF ROENTGEN-RAY PLATES.

PLATES I, II and III.—Three plates of the same skull, to show the effect of different positions of the X-ray tube. In Plate I the shadows of frontal sinuses are almost obscured by those of the base of the skull. In Plate II frontal and maxillary sinuses and ethmoid cells are shown. In Plate III the angle of the "principal ray" is too acute and there is too much distortion.

PLATE IV.—Frontal sinuses slightly asymmetrical. Left frontal and maxillary sinuses and ethmoid cells contain fluid.

PLATE V.—Maxillary sinuses and ethmoid cells normal. Frontal sinuses asymmetrical and slightly cloudy because very shallow.

DISCUSSION.

DR. JOHNSTON, Secretary of the American Roentgen Ray Society, emphasized the importance of X-ray diagnosis of sinusitis. It was easy enough to determine the presence or absence of fluid without this aid, but it was of value in the determination of the exact relationship of the fluid. Frontal sinus work was the most difficult he had ever tried to do, and Dr. Caldwell was easily the pioneer in this line. The failure of others was not due to the fact that Dr. Caldwell

possessed any particular secrets in the matter; the excellent results which he obtained were due to the refinement of technique, to his attention to infinite detail. Many operators failed to get satisfactory results because the patient was not properly immobilized. This, however, had been obviated by an apparatus devised for holding the person still. The plate was placed on the face and in contact with the frontal sinus, unless this was rendered impossible by a prominent nose, when it was placed as close as possible. The tube must be very efficient, must give off no secondary rays, and it must keep its vacuum through a long exposure. The development of the plates was that of any other plate, requiring no special developer. The accuracy of the diagnosis depended upon the technique and experience of the man who does the work. He would not make a diagnosis from a plate unless he knew the man who made it. He sometimes hesitated to make diagnoses from his own plates, although they might be pictorially beautiful results. The work was exceedingly difficult, and anyone undertaking it must expect failure for a time.

Dr. B. R. Shurley, of Detroit, Mich., spoke of the excellent work in this direction of Dr. Preston M. Hickey, of Detroit, President of the American Roentgen Ray Society, with whom he had worked for some years. During that time, he had every sinus case examined by this method, and had found the interpretation of the plates very interesting in connection with all other methods of diagnosis and particularly valuable in determining the outline and size of frontal sinus. It was also of value in ethmoidal disease where the diagnosis is doubtful.

Dr. Thomas J. Harris, of New York City, asked Dr. Caldwell to explain, in closing the discussion, what faults in technique accounted for the fact that, despite the perfect fulfilling of all the conditions specified by the speakers, the sinus would sometimes come out so poorly that one would hesitate to make a diagnosis from such plates.

Dr. Joseph Beck, of Chicago, said that he had had excellent results since he had begun to follow Dr. Caldwell's method. He had found it interesting and instructive to study the plates of the frontal sinuses, particularly where external operation was to be employed. The ethmoidal cells would show fairly well, but the antra were not mentioned by Dr. Caldwell. He asked how radiographs of these could be obtained.

Professor Jansen, in discussing Dr. Caldwell's paper, said: "I have been using for the past nine months a large apparatus, without an interrupter. The exposure is usually 2 1-2 to 3 1-2 minutes. I show you several photographs, which show the details of the temporal bone, beautifully; the horizontal and the semi-circular canals, the sigmoid sinus, and the cells of the mastoid process. In the case of the accessory sinuses, I always make three exposures, a frontal, and one profile view from either side. Through the two profile views I want to be able to compare the two frontal sinuses, ethmoidal cells, and sphenoidal sinuses, in regard to shadows; and also to determine the size of each frontal sinus, and to be protected against such surprises, as in these photographs, which I show you, in which the right frontal sinus is seen upon the left plate, and the left sinus upon the right plate. We are dealing in this case with a patient in whom one frontal sinus was operated upon several years ago."

Dr. Caldwell, in closing the discussion, said in answer to Dr. Harris's question that when the conditions specified have been perfectly fulfilled, good plates would practically always result. It was impossible, however, for the majority of operators to fulfill perfectly all of these conditions in every exposure. Sometimes when good plates were obtained a diagnosis from them is impossible because of some peculiarity in the development of the bones of the individual. He congratulated Dr. Shurley on having been able to avail himself of the efficient help of Dr. Hickey, whose splendid work was well known to the author. In answer to the question of Dr. Beck, Dr. Caldwell said the maxillary sinuses should be shown fairly well in the projection which is best adapted for showing the frontal sinuses. They were, perhaps, better shown by adopting the direction of rays commonly used by the German workers. The radiograph of the maxillary sinuses was usually considered of secondary importance, since their condition can be so easily determined by other means. With reference to the loss of hair, mentioned by Dr. Sprague, this was not likely to occur if, between the tube and the scalp, is inserted a filter consisting of a thin sheet of aluminium or, as proposed by Dr. Pfahler, a piece of sole leather. These unfortunate accidents seldom occurred except as the result of repeated exposures, and they served to emphasize the importance of strictest attention to technique, so that repeated exposures will be unnecessary in order to obtain a successful plate.

PAPER:

MALIGNANT DISEASE OF THE NOSE.

EDGAR M. HOLMES, M. D.

Occurring in the nose, a cavity so labyrinthine in structure, and even with present methods of examination so hidden from view in many of its recesses, malignant growth is frequently given an opportunity for considerable development and extension before the symptoms may be sufficient to call attention to the condition; and although the cases I report may offer nothing new, either in diagnosis, prognosis or treatment, they may, I hope, be of some service when added to many other recorded cases.

Whenever a malignant growth develops, the sooner it is removed the greater are the chances of eradicating it, and when there is suspicion of any growth being malignant the pathologist should be immediately called to our aid. In a general way, any vascular, more or less granular growth occurring in any locality within the nose should receive our immediate attention.

The symptoms associated with the early development of malignant nasal growth are found associated with many other less serious conditions. Hemorrhage may be an early symptom. General symptoms of rhinitis, as stuffiness, excessive secretion, and feeling of distention may be the only symptoms. Pain is not an early symptom, but was present in one of my cases.

When we have made a positive diagnosis of malignant growth in this region, it is often not an easy matter to decide whether or not to interfere, how extensively to sacrifice the surrounding tissue when we decide to operate, and how to alleviate, so far as possible, the suffering while the patient has to survive. Knowing that one has a fatal disease is none too pleasant, but when it is known that that disease may produce intense pain, affect sight and smell, and inflict an almost unbearable odor, it is a condition which demands any alleviation within our power to give. If only breathing space

can be for a time re-established, and pain and pressure reduced, it may be right to operate, and I find that most people prefer to submit to an operation when that is the only improvement which can be promised.

I think we all, who have to deal with malignant growths in this region, as well as those who have to meet similar conditions in other almost inoperable places, long for some form of treatment, serum, opsonic, or other, which will enable us to meet and conquer this terrible condition; but until then we must do all possible within our power to give relief.

As the locality of the disease may not only modify the treatment, operative or other, but also very materially affect the prognosis, I shall, as a matter of convenience, place the cases I am reporting in three classes. In the first class, we will consider those in which the growth is attached to the septum; in the second class, those in which the disease is restricted to the turbinal bodies; and in the third class, those cases which apparently have their origin in the accessory cavities connected with the nasal chambers. The pathological reports again classify our cases, but as I have so few to report I shall not try to thus classify them, except to give pathological records.

Of the eleven cases upon which I have operated—

Two were attached to septum.

Three were attached to middle turbinate.

One was attached to extreme anterior end of lower turbinate.

Three had their origin in Highmore's antrum, and two were so extensive when examined that I had no idea of their origin.

The pathologist reported both septal cases as carcinoma, one of the turbinal cases as epithelioma, and the other two as sarcoma, the lower turbinal case as epithelioma, the antral cases, one as carcinoma, another as enchondroma, and the third as carcinoma with areas resembling odontoma, the other two cases as adenoma and sarcoma.

The youngest patient was 17 years, and the eldest 70 years of age. The average age was 45½ years. Five were males and six were females. Three had lost near relatives with malignant growth. The others gave no such history. There was nothing about employment or habits which gave any suggestion as to etiology.

Of these cases two only are now living, and one of these, Mr. J. McN., came to me 12 years ago. He was a well developed and

healthy appearing man, 46 years of age, a salesman by occupation, and with a family and personal history of almost perfect health. He consulted me on account of nasal hemorrhage which was and had been his only symptom of disease. The cause of his hemorrhage was found to be a tumor about half an inch in diameter attached to the right side of the septum, at about the junction of the cartilage and the vomer, perhaps a little more over the vomer than the cartilage. The attachment was by a pedicel about one-fourth inch in diameter. The macroscopic appearance was that of an irregular nodular, bluish red colored polyp, which bled profusely after being moved by a probe.

I removed the mass with cold snare and cauterized the base very thoroughly with electric cautery. As the pathological report was carcinoma I kept close watch of patient and at the end of four weeks there were signs of similar tissue springing from the original site. I advised removing all of the septum at this area, including about one-third inch of apparently healthy tissue. This was consented to by the patient, and under cocaine with cartilage knife and scissors I removed the cartilage, bone and mucous membrane covering in one piece. This produced a very large perforation but it was so far back that there was sufficient cartilage anteriorly to maintain the proper contour of the nose. The pathological report upon the excised portion of septum was that the tissue at circumference was normal but at site of trouble there were areas of carcinomatous tissue, and also simple granulation tissue. This man has remained in perfect health since and this may give some hope in cases of malignant growth occuring in the nose, if so situated and of such extent that they can be removed entirely.

A case which belongs to this class, so far as symptoms, diagnosis, and treatment are concerned, was that of Mrs. T., 47 years of age. She was a widow with moderate means and though well nourished had a rather careworn appearance. She had been troubled with epistaxis for about six months. She was referred to me Nov. 27, 1897, by her family physician, Dr. Geo. E. Thompson, for an attack much more severe and of several hours' duration. The hemorrhage was from the right nostril and after stopping the flow of blood with a cotton plug I found that it was coming from a tumor about the size of a pea, rather nodular and vascular, attached to the septum anteriorly at about the location so often found irritated and abraded by picking the nose.

I removed the growth with cutting forceps and cauterized base with electric cautery. The excised portion was reported by the pathologist to be carcinoma. I requested patient to visit me for inspection but she did not come again until 5 weeks later when there was again hemorrhage. At this time at the posterior border of the operative area was a bluish red tumor about the size of an apple seed. I removed the septum for about a quarter of an inch about the diseased area, removing slightly more cartilage than perichondrium and mucous membrane, so that these would cover cartilaginous border of perforation made. There was comparatively rapid healing and no return of the growth at the end of 14 months, the last time I saw her. At about this time she removed to Maine where she died 18 months later of what was diagnosed as carcinoma of the uterus. There were no symptoms of any nasal disease during this period.

The next case is in a class much more formidable, due to location of origin, and extent of involvement. Mr. C. was referred to me by his family physician, Dr. Frame of Rockland, Mass., for a bleeding polyp of the right side of nose. He was a well developed and nourished man of 37 years of age, and his only symptoms were stuffing and bleeding of the right side of the nose, and some pain neuralgic in character over right side of head and at occiput. Nasal inspection showed a livid colored growth filling nares from about one-half inch inside anteriorly to about one-third inch from posterior choana. The least probing was followed by profuse hemorrhage. I removed piece of anterior end about size of hazel-nut for examination and the report from pathologist was that we had a spindle-celled sarcoma. Hoping that perhaps the attachment might be to middle turbinate and nowhere else, I advised immediate operation. The patient dreaded to undergo the same and deferred operation for about three weeks, when the pain became so severe that he sought any relief. At this time the growth had appeared at anterior nares, and there was distinct exophthalmos of the right eye. Under ether I removed the greater mass of growth with middle turbinate, but I found that it had filled ethmoid, and had pushed through cribriform plate. The case was of course hopeless, and I did not try to do more. Fortunately there was only one more week of suffering. This case illustrates how rapidly these growths can advance and how soon they may get beyond relief.

Case second in this class is Mr. L., who was referred to me by Dr. Tirrell of So. Weymouth, Dec. 7, 1897. Mr. L. was 62 years of age, was a wiry, rather small and somewhat anaemic man. He had always led an out-of-door life. His father died of pulmonary tuberculosis, his mother of cancer. There were then three living brothers, and one brother and one sister dead.

He had been in perfect health until about three years before consultation. The first symptoms were those of a cold in the head.

This symptom in various intensity had remained and the last weeks there had been pain. At first there was a pressure as of something pushing on nose, but lately considerable pain in nose and right eye. The right side of nose was pushed outward and a vascular nodular tumor apparently attached to right middle turbinated area filled the whole right nasal chamber. This bled very easily and there was considerable hemorrhage following a careful use of the probe.

Five days later under ether I removed with a hooked knife the middle turbinate with the attached tumor which was fully the size of a pigeon's egg. The inner wall of antrum had been pushed outward as had also the orbital plate. A specimen of this growth was examined at the pathological department of the Boston City Hospital, and was found to be carcinoma. This man has had no recurrence and Dr. Tirrell reports him as a healthy man to-day, 72 years of age.

The third case in this class was a Hebrew girl 17 years of age. She first came to me complaining of some pain in the right side of the head and stopping of the right side of the nose. She was at this time fairly well nourished and developed but she said she had lost considerable weight.

Under cocaine, anterior inspection of the nose showed a polyp-like but vascular mass, apparently in the naso-pharynx. This was shown by post-nasal mirror to practically fill the right side of the naso-pharynx.

The following day I removed the tumor with cold snare and cauterized the base with electric cautery. The growth was rather nodular and vascular and about three-fourths of an inch in long diameter and one-half inch in other. The pathological report was that we had a myxofibroma, and not a sarcoma as we had thought. In three weeks there was another growth in all appearance like

the first. I removed this together with the posterior two-thirds of the turbinal body.

The pathological examination of this specimen showed myxomatous and granulation tissue.

About four weeks later patient consulted me at my office as I was not then on duty at the City Hospital. There was now exophthalmos and complete blocking of the right side of the nose and naso-pharynx. I referred her back to the hospital and Dr. Leland removed a specimen for examination. This time it was reported that she had a rapidly growing spindle-cell sarcoma. The patient was admitted to hospital and Dr. Munro performed the Cheever operation, followed by death the next day. An autopsy could not be obtained but the operation showed that the process had extended into antrum, ethmoid, sphenoid and orbit.

John W., a robust man of 51, with an excellent family history and a perfect personal history except for children's diseases, consulted me February 11, 1903, for a painful and obstructive growth in anterior portion of nose. This he says to his knowledge he had had for about a year, but pain had been present about three weeks.

Examination showed a greyish red irregularly shaped tumor about 1-3x1-8 inches attached to anterior end of the right lower turbinate. The surface towards the septum was abraded and showed signs of ulceration. This was classified by the pathological department at the City Hospital as carcinoma. Under cocaine I removed the growth with considerable of the surrounding tissue. I adjusted and sutured edges of wound but on second day they pulled apart. The wound, however, was entirely healed at the end of the month. About three months after operation the patient began to have pain, and what he described as a sensation of pressure in the head. He slept poorly and lost weight rapidly. His symptoms increased and he finally died at the end of four months, just seven months after operation. Dr. Mahon, who saw patient the last few weeks of his life, reported that there was finally paralysis of whole left of the body and almost total blindness before death. Relatives refused autopsy so we can only reason by symptoms that there was probably extension into cranial cavity through the lymphatic or venous channels.

On Oct. 3, 1903, Mr. J. C. M. consulted me for a severe neuralgic pain in right cheek and eye. This had existed for five weeks but was increasing in severity and frequency of attacks. For

the few days previous to examination, the pain had been practically constant. Mr. M. was 53 years of age, weighed some 220 pounds and had the appearance of one who had been suffering. His pulse was 108 and his temperature was normal. His history, family and personal, was excellent. The right eye was apparently slightly prominent. The conjunctiva was injected. The vision was normal. On the right side of nose the mucous membrane was much inflamed, and there was considerable pus in the middle fossa. The right antrum was much more opaque than the left. After passing trochar I tried to cleanse antral cavity, but very little solution came from the ostium. A probe passed through trochar gave the sense of entering some pulpy material. Radical operation was advised but patient declared he would die before he would take ether. I saw him again two weeks later and at this time there was no doubt about the exophthalmos. There was a brownish red polyp in the middle fossa. A small piece of polyp was removed for examination and found to be carcinoma. The condition became rapidly worse and at end of four weeks there was total blindness which lasted nine days, when there was sudden death. There was no examination of the eyes after my second examination and yet I believe the carcinoma had extended into orbit and probably into cranial cavity.

On Oct. 11, 1906, Miss L. H. consulted me on account of nasal pressure and inability to breathe through nose. She was 30 years of age and poorly nourished, of a sallow complexion and a constant mouth breather. The first important fact in her past history was stuffing and tinnitus in left ear two years before. There had been plugging of nose about the same time. This had grown worse and about a year before consulting me she had been operated upon under ether at some hospital in New York. She could not tell the name of hospital or the surgeon who operated upon her.

Examination showed the right side of the nose to be filled with pus and polypoid tissue. The left side contained much thick foul pus and a very vascular kidney colored polypoid growth. A similar growth filled the naso-pharynx, pushing the left side of the soft palate forward. There was a small cicatrix in the left canine fossa.

I advised the patient to go to the hospital where I could operate upon her immediately, but this she did not do. On November 22, over five weeks later, she again came to my office. There

was now left exophthalmos and considerable pain, and she was willing to undergo an operation. The disease seemed so extensive I did not give a very good prognosis, and she did not appear at St. Elizabeth's Hospital until December 7th. I now found her to be suffering much pain. She had lost much weight, the pulse was weak and there was slight elevation of temperature. She was now determined to have something done. Under ether I made incision under the left lip from anterior border of canine fossa as far back as the second molar, the cheek was dissected to above the zygoma and to the infra-orbital foramen and what was left of the necrotic anterior wall of the antrum was removed. Through this opening was removed the growth which filled the antrum, the ethmoid, and the sphenoid cells, the posterior portion of left orbit as well as the whole left nasal and naso-pharyngeal cavities. The patient never thoroughly recovered from the ether, and died the night following the operation.

Dr. Leary who examined the growth reported it to be an endothelioma.

The third in this class was Rev. Dr. P., 70 years of age. He was referred to me by Dr. Darling for nasal obstruction and discharge from the left side of the nose. I first examined him March 18, 1907. He was a well preserved man for one of his years and except for prostatic trouble had been in good general health. Examination showed a very vascular pulpy growth about size of a cherry and apparently attached between the middle and the lower turbinate at usual ostium of the antrum. Trans-illumination showed much obstruction to light on the left side.

I advised immediate operation, fearing malignancy and hoping by performing a radical operation upon the antrum to perhaps eradicate the disease. As the patient had much church work which he had promised to perform, he put off operation until June 6th, when the tumor nearly filled nose and naso-pharynx. I was able by removing lateral wall of antrum to clear out that cavity, and then to remove through naso-pharynx the detached growth, which was the size of a goose egg. Dr. Mallory reported tumor to be carcinoma with areas of odontoma. So far as possible I removed all infected tissue and after the operation the patient quickly rallied and had no symptoms until the last of August, when there was again a very rapid development of the growth. As there was much distress from this second growth and as the patient very

much wished a few more weeks of life to complete certain work he desired any possible relief, and Sept. 5, he was again operated upon. There were after last operation several weeks of comfort. In November there were signs of a return of the disease and death came December 16, 1907.

Mrs. L. G., a poorly nourished, anaemic woman, 34 years of age, consulted me Jan. 8, 1901. Her father was living, her mother died of hemorrhage from the uterus. Her first symptoms began eight years before when she had trouble about breathing through right side of nose; there was also considerable nasal secretion. Her family physician had removed considerable flesh from the right side of the nose which he said was a polyp. Several times there had been operations upon the nose. These gave temporary relief. The last operation was about four months before coming to me. At this time there had been profuse hemorrhage and very little was accomplished. There had been much loss of weight and strength during the previous year.

The right side of the nose was filled with a brownish bluish red mass which had extended into the naso-pharynx sufficiently to block the left side.

Under cocaine I removed much of the mass from the right side of the nose and the entire mass from the naso-pharynx. The growth had entered or was in the antrum, filled the attic, was attached to lower turbinate and it was impossible to learn its origin. Pathologically it was round-cell sarcoma.

As patient lived in a distant village I did not see her again but a sister wrote me the following: "My sister was given much relief by your operation for about eleven or twelve weeks when there was a recurrence of her old symptoms. She grew quite rapidly worse and died Nov. 19, 1901. After she found she had cancer she said she would try to do no more."

This case was likely one of simple polyps at first and was operated upon several times for that condition. There seems no way of judging whether there was any connection between these and the first tumor which was sarcomatous.

Mrs. E. M. G., a well nourished and healthy appearing woman, 53 years of age, consulted me March 7, 1904. Her mother died of cancer of breast at 61 years of age. One sister died of uterine hemorrhage at about 46 years of age. Two brothers and three sisters were living and in good health.

Mrs. G. was the mother of three healthy children and had always been in excellent health herself until about one year before consulting me. Her first symptoms were those of simple rhinitis. These gradually increased until at end of five months she consulted a physician in Philadelphia. He told her she had a bad ozaena and gave some oily spray which she used for about three months. The symptoms gradually increased, there was inability to breathe through the nose most of the time, and at times a severe heavy pain over the eyes and at occiput.

For ten days there had been more or less nasal hemorrhage.

Examination showed a liver colored nodular growth filling both nares and pressing soft palate forward. The lower edge was visible in pharynx below palate.

I removed a section for pathological examination and Dr. Mallory reported it to be adenoma. There was such an extension of disease that prognosis was exceedingly poor, but patient wished to be relieved of nasal obstruction if but for a short time.

Under ether the nasal cavities and the accessory cells were cleared so far as possible but the growth had filled every space.

For six weeks patient was very comfortable when the right side of the nose became plugged. There were a few hemorrhages, but not of severe type until July 1, when there was a severe hemorrhage from mouth, and death followed before surgical aid arrived.

The study of these few cases shows how rapidly these nasal growths, whether carcinoma, endothelioma, or sarcoma, may progress, how they push into and fill the labyrinth of sponge-like cells of the ethmoid and the sphenoid, how liable they are to break down the base and enter the floor of the cranial cavity, and how extensive they are liable to be when first the patient consults a physician.

In preparing these reports I have been surprised to find how frequently growths in the nose which I feared might be malignant from the general appearance were reported to be benign by the pathologist.

PRESENTATION OF INSTRUMENTS.

Dr. Richards' Tonsil Forceps.

Dr. George L. Richards, of Fall River, Mass., presented tonsil forceps, in two sizes, devised by himself. He said that for a good many years those who have done work on the tonsils have felt the need of forceps which would grasp the tonsil without tearing, at the same time allowing one to place over it any preferred type of cutting instrument. These indications were met in the instrument presented. The forceps lock as soon as the tonsil is grasped and the dissection may be made in any manner desired, it being necessary to detach the forceps when the cutting instrument is applied. The smaller size was designed for the removal of small parts of the tonsil which might be left. He had used the instrument for six months before having the final model made, and had found it very satisfactory.

Modified Wright Snare.

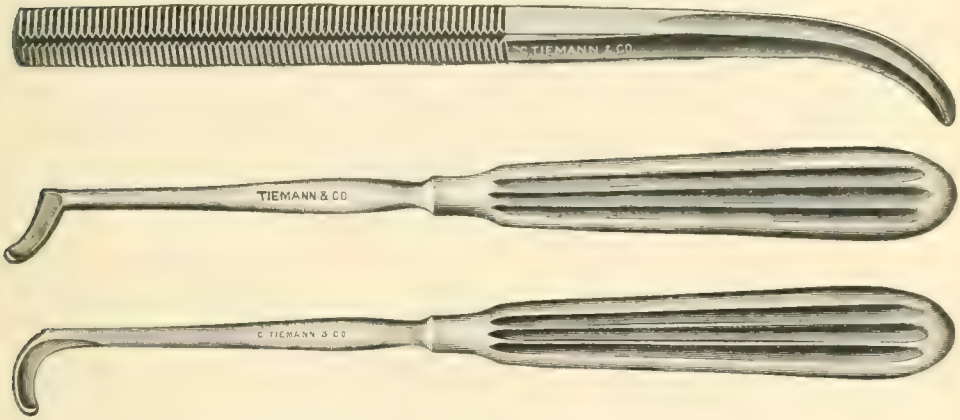
Dr. William C. Bane, of Denver, Colo., in presenting this instrument said that for a number of years he had used the Wright snare for nasal work. Finding it too light for the average tonsil, he had had a larger and heavier instrument on the same principle. It had proven very satisfactory in his hands. The Andrews vulsellum forceps were well adapted for the firm tonsil, but in closing it on a soft, friable tonsil it was apt to tear out. He had had the ratchet lengthened so as to permit of moderate pressure on any sized tonsil. The rongeur forceps presented were made for him several years ago, but the present model was slightly heavier than the original one. The shape and size of the instrument rendered it a valuable rongeur for mastoid work.

The rongeur is made by F. A. Hardy & Co., of Chicago.

Dr. Curtis' Antrum Gouges and Knives.

Dr. H. Holbrook Curtis, of New York City, presented two antrum gouges and two antrum knives employed by him for internal antrum

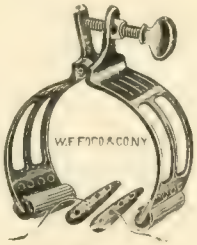
work. The gouges were very heavy, and with the knives one could do a very rapid operation. He had performed the operation very frequently by means of these instruments without a general anesthet-



ic by injecting the antrum with cocain and adrenalin, previous to the removal of the inferior turbinate and inner wall.

A Combined Bridge and Intranasal Splint for the Prevention and Correction of Depressed Deformities of the Nose.

Dr. William Wesley Carter, of New York City, presented the following apparatus as intended to fill a long felt want namely, that of a splint that would hold the nasal tissues in their proper position until union had occurred after fracture, both accidental and operative. It was especially adapted for the prevention and cure of depression of the bridge but it was also to be used whenever a broken nose needed to be held in normal position until union took place.

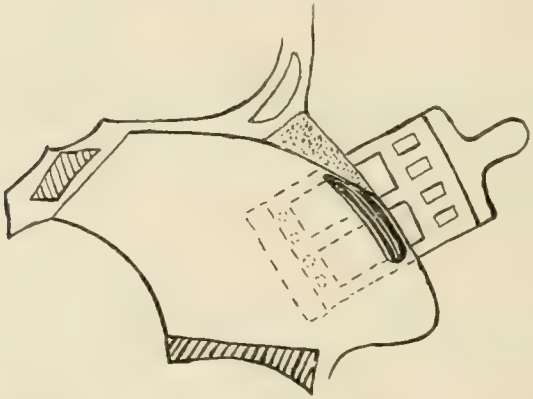


The instrument consisted of a bridge made of steel and as light as is compatible with strength. The bridge was formed by two

curved, fenestrated wings, 2½ cm wide, and joined by a hinge. The distance that these wings could be separated was regulated by a thumb-screw. The edges of the wings diverged to correspond to the shape of the nose, they were padded with rubber and near the margins were small holes that permitted of gauze padding being stitched on.

The second part of the apparatus consisted of two narrow hard-rubber splints, perforated by four small holes.

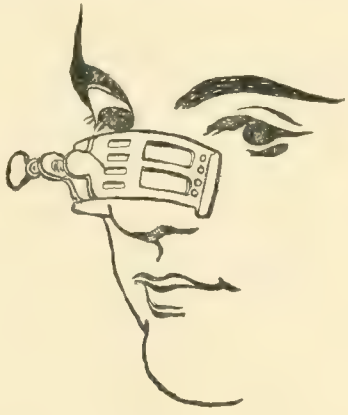
Assuming that the nasal tissues had been thoroughly mobilized, the application of the apparatus was as follows:



No. 14 iron-dyed silk was threaded through one of the holes in the small intra-nasal splint and knotted, (the hole was selected according to the distribution of lifting force desired) the other end was threaded into a large curved needle, this was passed from within the nose through the cartilaginous dorsum near its attachment to the nasal bones and close to the septum. The roof of the nose was very thin and easily perforated. The splint was now drawn into the nose and properly adjusted to the dorsum.

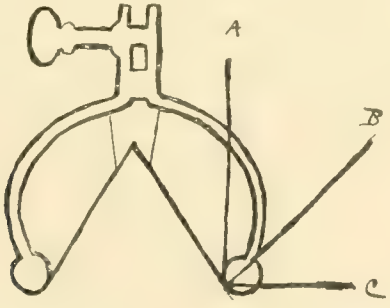
This process was repeated on the other side. The bridge was then fitted over the nose and the distance between the wings so adjusted by means of the thumb-screw as to give the proper support to the base of the nasal triangle. The sutures were now passed through the fenestrae of the bridge vertically above their exit from the nose and drawn tight enough to lift the dorsum into its proper position and reduce the deformity. The sutures were then tied together over the hinge of the bridge. There should only be sufficient tension to support the parts. The apparatus was auto-

matically held in place and there was no tendency to displacement. The splint should be worn from ten days to two weeks and the patient should be confined to bed during this time.



In uncomplicated fractures of the nose there was fixation in eight days and bony union in a fortnight.

The mechanics of this apparatus might be illustrated diagramatically: The line vertical of the base of the nasal triangle (a) represented the downward pressure of the bridge, produced by the tension of the sutures on the dorsum. The horizontal line (c) represented the pressure exerted horizontally by the wings and con-

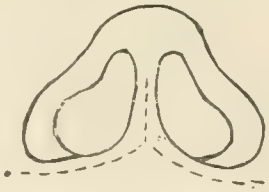


trolled by the thumb-screw. The mean direction of the pressure (b) therefore that is actually applied to the base of the nasal triangle bisects the angle formed by the vertical and horizontal lines, and gives us the proper support at the base, while the lifting force is applied to the apex through the sutures attached to the intra-nasal splints.

The combined action of these forces therefore would tend to construct a symmetrical nose.

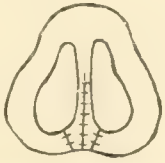
In nearly all cases of flat nose the septum was too short to admit of raising the bridge. To overcome this difficulty he had devised the following operation for lengthening it without leaving a perforation.

Through a button-hole split he elevated the mucous membrane on one side of the septum. With a sharp pointed bistoury introduced into this subperichondrial pouch he divided, beginning at the anterior edge of the vertical plate of the ethmoid, the cartilage and mucous membrane of the other side at a high level. (a) He then divided the mucous membrane of the remaining side at a much



lower level, (b) thus leaving a long flap of mucous membrane attached to the upper segment of the septum, when the latter was reached, this flap fell into the interval and prevented a perforation. The fore part of the septum was lengthened in the following manner: Downward from the septum an inverted Y-shaped in-

cision in the floor of either nostril was then extended under the floor of either nostril, making two flaps which were brought together in the median line and sutured with fine silk thus lengthening the septum and raising the tip of the nose. The incision in the floor of either nostril was then extended under the alae liberating them from the underlying tissue so that they could be brought toward the median line and sutured thus narrowing the nostril and producing a shapely nasal orifice.



The combined bridge and intranasal splint had the advantage of restoring the function as well as the contour of the nose by a re-arrangement of its parts and without the introduction of foreign substances into its tissues. A combination of its lines of force would tend to construct a normal nasal arch; it reversed the action of the forces that produced the deformity.

Note: Since the above presentation, Dr. Carter's attention had been called to a mask devised by Dr. O. B. Douglas and which at a glance would seem similar to this instrument. It differed, however, considerably in mechanical construction and application from his, Dr. Carter's being adjustable and giving support to the base of the nasal triangle, a most important consideration.

MEETING OF SOUTHERN SECTION, HELD IN BALTIMORE, DECEMBER 27-28, 1907.

PAPER:

A PLEA FOR CONSERVATISM IN THE TREATMENT OF CERTAIN AFFECTIONS OF THE NOSE AND EAR.

HARRY L. MYERS, M. D.

Some one has aptly said, "It is better to be conservative in radicalism than radical in conservatism." The difficulty in this age of advanced surgery seems to be in the finding of a safe channel between the Scylla of conservatism and the Charybdis of radicalism. I fear that I shall not be able to point out the way in these troubled waters, but I hope to create a discussion at this meeting by which I may learn the better to avoid the dangerous rocks on either side. I come to learn, not to teach.

With greater experience I believe that there has arisen a spirit of conservatism among us which will continue to grow. The literature of the day confirms this and, recently, my courage to present this subject has been not a little strengthened by articles from some of our best men expressing their belief in conservative methods of treatment in diseases of the ear and nose. It seems to me that it is in the surgical treatment of nasal diseases especially that more conservatism is needed. The antrum of Highmore, owing to its size, peculiar shape and dependent position is not only particularly liable to infection but must frequently suffer from involvement of the sinuses situated above it, collecting their secretions which pour down, thus sometimes acting as a reservoir and assuming the appearance of guilt when it is really innocent.

The operation upon this sinus through the inferior meatus marks, in my opinion, one of the greatest advances in the surgery of the nose and is really, compared to the external operation, a victory

for conservatism in that it is now used almost exclusively in work on this sinus except in those cases where there is sufficient necrosis to demand an external opening. I, however, think that even this operation, on account of its ease of performance and the lack of the necessity of general anesthesia in many cases, is apt to tempt us to destroy a large piece of the inferior turbinate and a goodly portion of the inner wall of the sinus when frequently we could accomplish a cure by puncture through the inner wall of the sinus under the turbinate and irrigation of the cavity every other day with a bland alkaline solution over a period of three weeks or two months. Many cases which have extended over months and whose discharges were decidedly offensive have yielded to these mild measures at my own hands. I do not mean to say that the cases I have been fortunate with were all cured by irrigation alone. When there is obstruction around the ostium of the sinus, whether it be from enlarged turbinate, deviated septum, polypi, or disease in the contiguous ethmoidal cells, I have removed these obstructions. In fact I may say that in nearly all except acute cases and in some of these I think we will have to, sooner or later, remove the anterior end of the middle turbinate. This is, however, a conservative measure and does not seem to do other than good. I am convinced that were more work done on the middle turbinate in these cases and less on the inferior, results would in the end be better. It has been pretty well proven that the inferior turbinate has a well defined place in the moisture producing function of the nose and I never could persuade myself that any more of it than absolutely necessary should be removed.

Before resorting to this operation we should be as certain as possible that the antrum is not acting simply as a reservoir, as under such circumstances we should fail to accomplish our end and at the same time would produce an abnormality to no purpose. In most cases of chronic discharge from the antrum there will be found disease in other sinuses above it and these sinuses should be treated and their secretion stopped if possible, before more than irrigation is done to the antrum. Those cases of antral discharge where the trouble can be traced to diseased teeth generally yield to drainage and irrigation through a generous opening in the tooth cavity after the offending member has been withdrawn. Greater patience, however, is required in these cases as

time must be given for nature to throw off the necrotic tissue which may have formed in the floor of the antrum, around the diseased root. I may say here that I have experienced no trouble with food getting in through the opening in the alveolus and I neither use an obturator nor a tube. I have experienced little trouble in the cavity closing as I direct the patient to use a Moffett's Dental Syringe which has a conical canula with a rather sharp point, thus being easy of introduction and serving to push back the part effectively without pain. Where there is much necrosis I believe the older operation from the outside is necessary.

We of the South have fewer cases of disease of the frontal sinuses with which to deal and I feel less competent to speak on this subject, but most cases which have come under my observation have been rendered comfortable and in many cases cured of pain and discharge by conservative measures within the nose. I find that the removal of the anterior end of the middle turbinate and other obstructions to the infundibular opening together with irrigation has been of such service that few of my patients were willing to have resort to the external operation. I have never tried the method of boring into this sinus from the nose and I never shall as I consider it impossible for any one, however skillful, to know where the end of his drill is going, and as between this method of treatment and that of an external opening, I think the external method more conservative. I think, however, one may accomplish much by the cautious use of the curette in enlarging the canal in an anterior direction as recommended by Hurd; where there is bulging of the eye or tissues or where pain is not relieved by conservative internal methods, the external operation often becomes a necessity. I recognize that I am treading on dangerous ground when I attack some of the methods used to-day in dealing with septal deviations, but I am afraid that we are sometimes too bold in the use of the "window" resection operation. First, let me say that I feel that within proper limits it is the greatest advance in nasal surgery and will eventually be the principal method used for corrections of septal deformity. To my mind the danger of it lies not in its use but its abuse. The more skillful we become in its technique the more anxious we are to remove every bump and projection; in other words we strive for a septum that looks perfect. In our enthusiasm for such a

result I believe we sometimes remove not only more tissue than is necessary but sometimes more than is safe. It seems to me that what we should strive for is to remove as little as possible in order to restore good nasal breathing regardless of appearance. We all know of very crooked septa where nasal breathing is perfect and a projection which does not obstruct should be left alone. I am also of the opinion that we cannot yet afford to give up the nasal saw, for with it we can many times accomplish as good a result and we certainly feel more certain of the contour of the external nose.

The treatment of hypertrophic rhinitis has made advances in late years due perhaps no little to an improved armamentarium and the established view that it rarely does harm to remove portions of the turbinate bodies with scissors and such instruments. The careless use of the galvano-cautery has been so criticised that we are in a fair way to lose a very good means of relief in mild cases of this trouble; a means that is being used today by men of undoubted experience and whose results are second to none. In those cases where there is no great thickening of the soft tissues but where the patient complains of great stuffiness at times and in cases of intumescent rhinitis where the patient either will not or cannot live out of doors, I have found linear burns along the inferior turbinate with either the galvanic or chemical cautery to give me most excellent results, and I do not believe our enthusiasm for the newer cutting methods should lead us to abandon entirely the more conservative method in selected cases. Where there is great hypertrophy or deformity the snare or scissors have given me better results, but even here I think as little as possible of the bony turbinate should be sacrificed, except where the middle turbinate is concerned, and here I believe the cautery should never be used.

In chronic suppurative diseases of the middle ear when to relinquish conservative measures for radical treatment, must necessarily depend upon several conditions. Environment for instance must be considered, for when a patient lives within easy reach of a competent aurist and has the means to obtain skillful treatment, he, in the absence of urgent symptoms, may frequently be made comfortable and rendered reasonably safe by the occasional use of intra-aural methods of treatment, and I am of the opinion that we are scarcely justified in operating upon such a case

radically when about the only symptom of annoyance is more or less discharge from the ear. On the other hand the patient who is suffering from a chronic aural discharge with occasional acute exacerbation who must live far removed from skillful treatment had best submit to the radical operation.

Another condition which would indicate the necessity for radicalism is an unconquerable offensive discharge which in some cases renders the life of the patient miserable. When the clearing out of the tympanic cavity from the meatus either with the curette, chemicals, syringe or the operation of ossiculectomy (which in my experience, while not always causing complete cure has never failed to improve) does not relieve pain and furnish good drainage and fails to greatly relieve all other symptoms, the radical operation should be considered. When the probe demonstrates necrotic bone in the attic and when cholesteatomatus material is present in the discharges, we must sooner or later resort to radical measures.

In a report some time ago recorded by Dr. Harris before this Society he showed that during ten years in one of New York's institutions for treatment of ear diseases, out of 32,000 odd cases of ear diseases there were only 60 cases of intra-cranial involvement. In the same report he showed from statistics of many authorities that intra-cranial involvement was very rare.

Koerner made the statement that the radical operation is indicated as soon as a diagnosis of chronic purulent otitis media is positively made but he also states that he has never seen or heard of a case of semi-mucopurulent discharge from the antrum, without obstruction to the outflow, that resulted in intra-cranial disease In my own experience extending over a period of thirteen years, I have seen meningitis once in a chronic case, serous meningitis once in an acute case, sinus thrombosis five times in acute cases and twice in chronic cases. Since intra-cranial complications seem to be rare I have felt that we should employ every possible conservative means of relieving these cases before resorting to radical work which often not only fails to cure, but too often renders the patient's life more miserable.

PAPER:

AN UNRECOGNIZED FOREIGN BODY IN THE LARYNX.

EMILIO MARTINEZ, M. D.

Errors in diagnosis are many times the source of instruction and perhaps more so than the study of successful cases. Without any desire to criticise the work of the able laryngologists who handled this case before I did and without any attempt to excuse my own error, I think that this clinical history is not devoid of interest.

Near the end of February last, a young man named Adolph L., a native of Cuba, twenty-five years of age, mechanic, consulted me, making the following statement:

On the night of July 18th, 1905, while living in Brooklyn, N. Y., he had the misfortune to swallow his tooth plate. Dr. B., who attended him at first, tried to extract it but found it impossible and took him to a hospital nearby, where another trial was made under chloroform, also without result. Next day a radiograph was taken and he was sent to another hospital in Brooklyn, and esophagotomy was performed on the afternoon of the twentieth, and the plate safely extracted. He remained in the hospital for a month. Near the end of September he was able to work at his trade as a mechanic, and went to his shop; but he noticed that his respiration was short and he suffered several attacks of asphyxia. He applied again to Dr. B., who performed tracheotomy on the ninth of October. Following this operaton his larynx was treated, several incisions were made and intubation was attempted twice, but he could not retain the tube. Nineteen days after tracheotomy the canula was removed, but had to be replaced; four days later he was discharged from the hospital. Dr. B. treated him at his office up to the middle of December, without any benefit. A friend took him to one of the large New York dispensaries, which he attended regularly and was assisted by prominent specialists. He states that he was considered "an interesting case," that many doctors examined him and he

served as a subject for various lectures before a large class. He was treated all this time with high doses of iodide of potassium. The professors of this dispensary refused to withdraw the canula and sent him back to his doctor. Dr. B. advised him to wait seven or eight months to have his canula removed, but as he had to live by his work and he could not do it as a mechanic in this condition, he resolved to return to Cuba and sailed on the eighth of February.

When I saw this young man on his arrival on the 12th of February of 1900, he had a complete paralysis of the left vocal cord, evidently from injury to the left inferior laryngeal nerve during the esophagotomy, and marked laryngeal stenosis, not only from the paralysis of the cord, but also from the swelling of the right ventricular band, which occluded the cord on that side. There was apparently no growth on the surface, because the mucous membrane was normal, but the movements were impaired. The patient was hoarse, although he could make himself understood with the tracheal tube closed. A slight cough, every now and then, brought up some secretion but otherwise he was not troubled by his larynx, there being no disphagia, or pain whatever. His general health was excellent.

I did not make a diagnosis, but was inclined to accept the possibility of syphilis of the larynx, as the New York specialists had done, although any antecedent in this respect was denied. He went to his home at Matanzas and two weeks later wrote to me that Dr. O. of the Matanzas Civil Hospital was disposed to withdraw the canula if I advised it. I made no objection, provided he remained at the hospital for some time to avoid an accident. The canula was taken out, and he breathed tolerably well, although he could do no hard work. He remained with his family at Matanzas up to October of the same year, returning at that time to Havana with increased difficulty in respiration, which subsided after treatment with steam inhalations. In December, he once more returned with an attack of asphyxia and I sent him to the Mercedes Hospital of Havana, and my assistant, Dr. Hernando, performed tracheotomy again. He was discharged and returned home, but in March of this year he came back resolved to have something done because he could do no hard work with his canula in place.

I had discarded syphilis as a diagnosis by this time because iodide of potassium had no effect on the lesion; it was not a cicatricial post-syphilitic stenosis but an active subacute process. The possibility

of neoplasm was suggested by a prominent laryngologist of Havana, who examined the case with me, but the age of the patient and the long time the trouble had remained stationary, obliged me to abandon this diagnosis also; I then suspected there might be a perichondritis of the interior aspect of the thyroid plate, but really this had no better foundation than the preceding ones. I explained the situation to my patient and proposed an exploratory thyrotomy, requesting his permission to proceed then according to the conditions that I might find in the larynx. He willingly accepted and would submit to any risk, as life was a burden to him with this disease.

With a free hand to do as I might think proper, I opened his larynx on the 20th of March (1907), in the ordinary manner for thyrotomy, and after cutting through the cartilage I found the mucous membrane separated, forming a cavity which I supposed was the area of necrosed cartilage. Passing in a curette I commenced to scrape on a rough surface, and was very much pleased to have found a diagnosis for my patient, when suddenly the rough surface gave way and I brought it out on my curette. It was a flat bone with a rounded border and rough ends, evidently a fragment of a small rib, the patient supposes, a piece of mutton chop, which he was very fond of eating. The cavity was full of granulation tissue, but otherwise was smooth. After placing a catgut suture to readjust the thyroid, I closed the wound.

The patient made an excellent recovery, and two weeks later I removed the tracheal canula, there being no longer any stenosis and the man breathed freely through his larynx; a small flap of mucous membrane which protruded from the ventricle, I excised with a laryngeal guillotine. He has returned to his shop in Brooklyn, and on the ninth of November last he reported that he was in good condition.

Reviewing the case, I presume that the patient swallowed the bone some time in September, 1905. On account of the recurrent paralysis, he could not dislodge it. He told me that this often happened to him, at that time with grains of rice and other small food particles. The bone was forced into the right ventricle and formed a cavity for itself eroding the mucous membrane and remaining there, occluding the lumen for twenty months.

Two important conclusions may be derived from this case:

(a) The remarkable tolerance of the larynx towards a foreign body for so long a period as twenty months, without impairing the patient's health, without great pain and with the only inconvenience of obstructing the glottis by its bulk.

(b) The danger to which any person, with recurrent paralysis, is exposed by his inability to expel a foreign body falling in the cavity of the larynx.

PAPER:

RETENTION IN THE MAXILLARY ANTRUM OF AN IRON BOLT THREE-EIGHTHS OF AN INCH IN THICKNESS AND ONE AND FIVE-EIGHTHS INCHES IN LENGTH FOR A PERIOD OF FOUR YEARS WITHOUT IMPORTANT UNPLEASANT SYMPTOMS.

JNO. C. McREYNOLDS, M. S., M. D., LL. D.

The patient, Mr. W. M. C., an electrician, 36 years old, came with the following history:

In February, 1903, while romping with a boy, he was thrown forcibly against the lock of a screen door inflicting a wound along the infraorbital ridge, but without producing any disturbance whatever of the eye. The wound through the soft tissues was closed with four stitches by the family physician who did not apprehend that any injury had been sustained by the bony structures beneath.

There was some bleeding from the nose at the time of the accident, but no significance was attached to it. The external wound healed promptly by first intention and there never developed at any subsequent time any evidence of a purulent accumulation within the antrum. For a few weeks, two years after the injury the patient suffered somewhat with what he called facial neuralgia on the injured side, but it passed off completely without creating any suspicion of a foreign body.

After the accident it was discovered that the screen door lock had disappeared and a search was instituted for it, but without avail. About two years ago, the patient observed that the two posterior molar teeth of the upper jaw on the injured side were becoming loose, and this condition gradually became more marked until in October, 1907, more than four years after the injury, the patient with his fingers extracted the two loosened teeth and then discovered a hard foreign body presenting at the opening thus produced. After a considerable amount of speculation as to the nature of this unsuspected intruder the bolt was easily removed by the astonished patient, who was then able to explain for the first time the mysterious disappearance of the screen door lock after the violent collision several years ago.

The case is one of interest, as it illustrates the high degree of toleration of foreign bodies which the maxillary antrum may endure.

PAPER:

REPORT OF A REMARKABLE CASE OF SIGMOID SINUS THROMBOSIS WITH MULTIFORM SEPTICO-PYEMIC COMPLICATIONS EXTENDING OVER A PERIOD OF FOUR AND A HALF MONTHS. ULTIMATE DEATH.

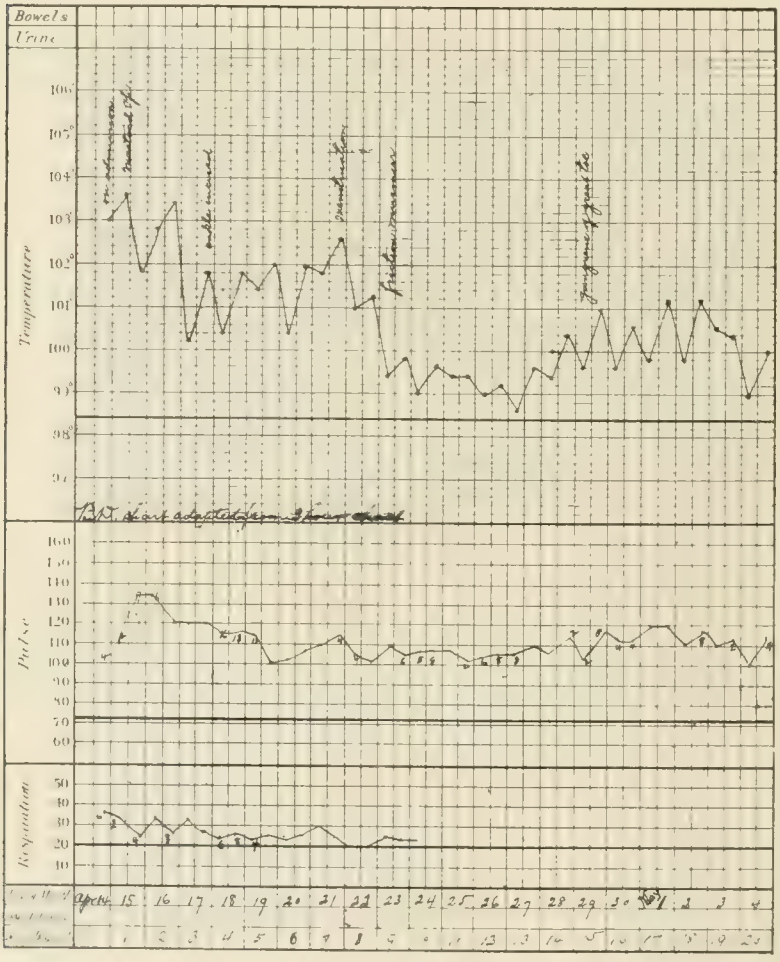
JOHN R. WINSLOW, M. D.

I was summoned, in conjunction with Professor Randolph Winslow, April 13th, 1901 (afternoon), by my friend Dr. Henry F. Hill, in consultation upon a school girl, aged seventeen years, presenting the following history:

Patient had scarlet fever when 7 or 8 years of age, followed by a discharging left ear; this was arrested by treatment and there had been no discharge for two or three years prior to this sickness.

Present attack began ten days previously, with sore throat, fever, cough and aural discharge; the patient did not seem ill, however, until April 12th, when the temperature registered 105.8°, pulse 120.

Upon examination the following day, I found the *left ear* stone deaf, but the shouting voice could be heard when almost in contact with the right ear. The left eye-lid was edematous (Stirling's



sign), but could be elevated; there was some exophthalmos (phlebitis of cavernous sinus?), but the movements of the globe were good. Patient could count fingers. No ophthalmoscopy.

Edema behind the mastoid and down the neck (Griesinger's sign), well marked. The bone itself was not very sensitive. Ten-

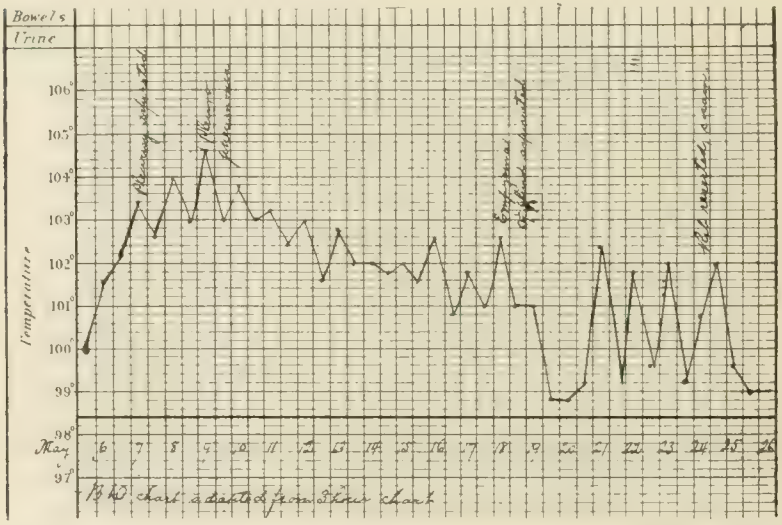
derness of upper posterior cervical triangle (Whiting's sign) absent. The face and neck were moist, but sweating was not profuse; no tenderness along the course of the internal jugular vein. Temperature 103°, pulse not recorded. The patient had had no chill, nor chilly sensations, no nausea, nor vomiting. The intellect was good, although she was drowsy and tremulous.

The nasal mucous membrane was intensely congested; *no aural discharge;* left posterior superior meatal wall bulged downward, concealing subjacent structures. Over this area, exquisite sensitiveness on probing. Having recently read Whiting's classical article upon the subject (Trans. Sec. Lar. A.M.A., 1899), although this was the first case that I had encountered, I had no hesitancy in diagnosing sinus thrombosis and urging immediate operation. Owing to reluctance on the part of the family to accept operation and questioning of diagnosis on the part of one of the consultants, it was decided to employ continuous hot bichloride irrigations, 1-5000, and to await developments. The following morning, April 14th, the patient manifested no improvement in other respects, and the additional symptom of a suspicious seizure early that morning resembling a convulsion. The significance of this will be discussed later on. The family and consultants then agreed upon operation. The house being infected with scarlatina and under quarantine, considerable delay was occasioned in obtaining a permit to remove the patient. She entered the University Hospital the evening of April 14th, temperature 103.6°, pulse 112, leucocytosis 14,400. I performed the Koerner radical mastoid operation, assisted by Drs. Randolph Winslow and E. J. Bernstein, April 15th at 2:30 p. m. The bone was very dense; a few granulations were found in the antrum but no pus. The dura was exposed in several locations and looked healthy. The sigmoid sinus was exposed from above the knee to near the jugular bulb. Its walls felt thick like rubber, but pulsations could be distinctly felt. The sinus was slit open with a bistoury under control of compresses above and below, and the blood in it was found fluid. A wire curette was inserted into the lumen and small black clots removed (streptococci). The condition was then one of peripheral thrombosis. The circulation was freely established both above and below, ligation of the jugular was not deemed necessary, and the patient's condition rendered it inadvisable. The wound was firmly packed, and, owing to the exposed dura and other complications, was only partially closed by suture.

Following operation, the evening temperature fell to 101.8°, which decline continued steadily until the morning of the third day, when it was 100.2°. The first complication appeared on the evening of the fourth day when the temperature rose to 101.8° and the right ankle was incised and found to contain pus (arthritis); this contained strept cocci. The temperature then remained between 101° and 102° until the seventh day, when a sudden rise to 103° was found to mark the onset of menstruation.

A rapid fall of temperature ensued, and on the tenth day it was 99°; at this period a friction murmur was heard over the left chest.

The temperature subsequently fluctuated between 99° and 100° until the evening of the fifteenth day, when a rise to 101° was



found to announce a new complication, gangrene of the end of the left great toe. This was amputated.

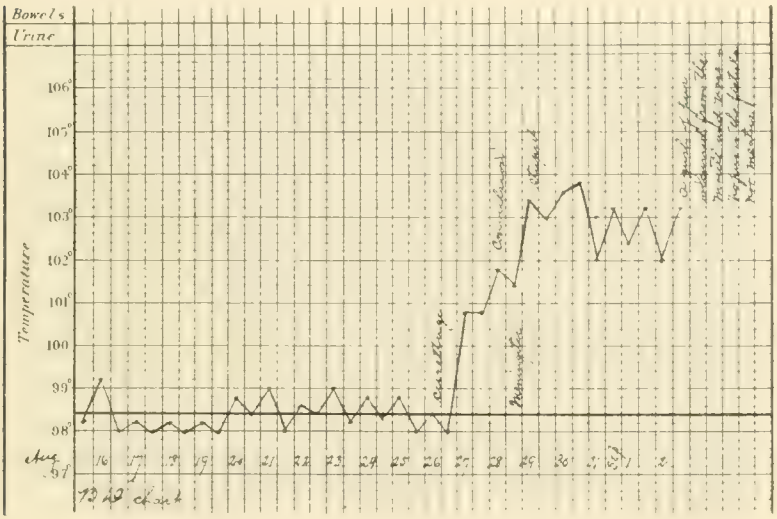
The temperature remained between 100° and 101° until the twenty-second day, when the gradual ascent began, which reached 103-4° on the twenty-third day. On this day the patient awakened with severe pain in the left side which was flat on percussion, and upon aspiration about one pint of serum was withdrawn from the left chest (pleurisy). Following this a slight fall of temperature occurred, succeeded by a rapid rise to 104° on the evening of the twenty-fourth day.

On the afternoon of the twenty-fifth day, the temperature was 104.6°, pulse 168, leucocytosis 28,000. Dry cough appeared and

persisted for many days thereafter as an harassing symptom.

A diagnosis of *septic pleuro-pneumonia* was made by the consultants, Drs. Randolph Winslow, Henry Hill and myself, and a fatal prognosis given. No one of us expected to find the patient alive the next morning.

Cold sponging was, however, instituted and stimulating hypodermic injections and enemata administered, and to the tireless faithfulness and efficiency of the nurse, Miss M. Miller, now superintendent of nurses at the Presbyterian Eye, Ear and Throat Hospital, in carrying out these instructions, was largely due the salvation of the patient.



On the morning of the twenty-sixth day, the temperature was 103°, pulse 156, leucocytosis 36,000; this very gradually fell to 102.4° on the twenty-eight day, and continued until the thirty-second day, when it began to fluctuate between 101° and 102°. On the thirty-fifth day about one pint of cloudy fluid was withdrawn from the chest by aspiration, which was found to contain pus cells and some streptococci (empyema).

From the thirty-second to the forty-first day a typical septic chart was presented, varying from 99° to 103°. Cough was very troublesome. At this time, it was thought imperative to establish better drainage of the chest, and Dr. Randolph Winslow *resected one of the ribs* under cocaine, as the patient's condition did not admit of general anesthesia.

The temperature promptly dropped from 102° on the morning of the forty-first day, to 99° the following morning, remaining around this point until the forty-seventh day.

The temperature then began to oscillate, sometimes being as low as 99° and sometimes as high as 101.8°, without special incident, until the patient was discharged from the Hospital on the eighty-second day (July 5th).

During this period *obstipation* was a noteworthy symptom and repeated doses of calomel and purgatives had to be administered.

From July 5th until August 20th, the temperature and pulse were quite variable; the greater part of the time the temperature was approximately normal for considerable periods. The patient was going out of doors, and was in such good condition that I seized this opoprtunity of securing a week's vacation at the seashore.

A significant fact during this period was the sudden occurrence on several occasions of elevated temperature, at times reaching 103° without apparent reason. Preceding the final illness there had been a period of twelve days of approximately normal temperature (98° to 99°).

Wound healing had progressed favorably and the side wound was well.

The mastoid wound was reduced to a fistulous tract, not larger than a slate pencil and about 3-4 inch in depth. This had become so obstructed by exuberant granulations, as to prevent drainage; and I accordingly curetted it lightly under cocaine *mot*, with the customary aseptic precautions (August 26th).

No exposed bone nor other complication was detected. The evening of the following day the temperature shot up to 100.8°, progressively rising to 104.8°, on the fifth day, and remaining around 103° until the fatal termination on the eighth day (September 2nd).

During this period there were typical signs of meningeal irritation, and on one occasion a convulsive seizure occurred. No pus was present in the wound nor in the meatus. At the time of death a gush of pus occurred from the *mouth* and from the *nose*.

Thus ended a heroic battle against death in many forms, during a period of four and a half months, and were blighted hopes of a surgical triumph. No autopsy could be obtained, but the fistula and the meatus were examined carefully and no pus found.

The following queries occurred to me at the time and have never been satisfactorily answered in my mind:

What was the exact cause of death?

What was the relation of the curettage to subsequent developments? As this was performed under strict aseptic conditions, how could reinfection occur? and even so, why should the phenomena of infection be manifested at so distant a point as the *anterior* cerebral regions?

Was a thrombus released? or were strepococci already present in the wound, stirred up to increased virulence?

Was the association merely a coincidence, and was there a latent brain abscess from the beginning? In this connection, what was the significance of the convulsiform seizures at the onset and the termination of the illness?

It should be here stated that I learned subsequently that the patient had told some friends just before her fatal illness that she felt well enough to be sure that she "left part in her head" since the beginning of her sickness."

This case has been selected for presentation from among several subsequent cases because it presents the following points of interest:

1. Classical symptoms and early diagnosis, in a condition of peripheral thrombus.

2. Multiform complications—purulent arthritis (toe and ankle) serous pleurisy, empyema (resection of rib), meningitis or brain abscess.

3. Desperate virulence of the infection (streptococci found in all thick [illegible] during [illegible] which made the patient [illegible] never really free from sepsis.

4. Obscure cause of death.

CANDIDATE'S THESIS.

A REPORT OF FIVE CASES OF THROMBOSIS OF THE LATERAL SINUS WITH RECOVERY, BEARING UPON THE DIAGNOSIS AND PROGNOSIS OF THIS AFFECTION.

HARRY FRIEDENWALD, M. D.

To those of us who as students learnt about thrombosis of the lateral sinus as a hopelessly fatal complication of suppurative otitis media, and who have followed the developments in its recognition and treatment, each case presents an exceedingly interesting problem. In June, 1900, I was able to report my first* successful operation, the first, I believe, in this city. The favorable termination alone of this and of several recent cases, while very gratifying, would not, however, justify me reporting them to this body. For the number of cases which have already been reported is very large. In the first edition of Koerner's monograph, Die Otitischen Erkrankungen des Gehirns, (1894) twenty cases were noted; in the second (1896) seventy-six cases, and in the third (1902) three hundred and eight are used as the basis of a statistical study. And the increase has gone on at the same rate. In making the following report it is my purpose not so much to put the cases on record, as to draw attention to certain important points bearing on the diagnosis and prognosis of thrombosis of the lateral sinus.

In the published case referred to, the patient was a man, aged twenty-nine, who had otitis media following facial erysipelas. The drum had been punctured without relieving the pain. After suffering with the ear for about two weeks he entered the City Hospital on January 6th, 1900. His temperature fluctuated between 99.8° and 101.2° until the evening of January 9th, when it rose,

*Maryland Medical Journal, June, 1900.

within a few hours, to 105.2° without any chill. On the following morning it was 100.5°. His mastoid was freely opened and the tympanic membrane incised. On January 11th the temperature rose to 104° (pulse 105), with a subsequent drop to 101° (and pulse 105) on the 12th, but again reached 104° (pulse 100) in the evening, without a chill at any time. During the following days there were fluctuations between 99.5° and 103° until January 17th, when 105.2° was again reached. The blood had been frequently examined and no leucocytosis found. This fact, together with the absence of chills and the great relief of pain after the operation, made the diagnosis of thrombosis very doubtful, but when the temperature again rose on the 17th to 105.2°, the diagnosis was made, and the operation for thrombosis without ligature of the jugular was performed. A broken-down, purulent thrombus was found shut in by a solid thrombus on both ends, the whole occupying about two and a half inches. It was thoroughly cleaned out. Cultures from the thrombus developed streptococci. In this case it will be seen that the mastoid operation was performed within twenty-four hours after the first very high temperature was noted, and the lateral sinus was opened eight days later. The patient made a perfect recovery. From this case I learnt that the severe chills upon which most writers had laid great stress may be entirely absent, and must not be regarded as a *sine qua non* for the diagnosis. The case also demonstrated that the absence of leucocytosis is likewise no reliable guide.

Case 2. Miss M. C., aged sixteen, a large, well-developed girl, came to my office on January 21, 1907, complaining of some aching in both ears, following grippe. The left drum was somewhat bulging and congested, the right slightly congested. A ten per cent solution of carbolic acid in glycerine was ordered to be dropped into the ears, and she returned on Jan. 23d, 25th and 30th, the pain having gradually ceased and the congestion rapidly diminishing. On February 2d I noted: "Left drum slightly congested, almost normal." She was apparently well until February 10th, when, after exposure in coasting in the country, the ear became very painful and she was sent into the City Hospital on February 12, where I saw her within a few hours. Her drumhead was greatly congested and bulging, she was suffering intensely, her temperature was 102° (pulse 112), and I incised the drumhead freely under ether anesthesia. The incision gave her great relief; she improved continu-

ously, her temperature fell to between 101° and 99.5° on the following day; on the third day it rose to 101.6° (pulse 100), but fell to 98°; and on the fourth day it reached 99.8° and fell to 98°. At this time the patient was feeling very well; the ear was discharging moderately. On the fifth day it rapidly rose to 104° (pulse 120) at midday. I saw her soon afterwards. The patient was stupid, but it was not difficult to rouse her. She complained of much headache. A blood count at this time gave a leucocyte count of 9,400. Between eight and nine, I saw the patient again. Her condition was unchanged; her temperature, which had fallen to 102.2°, was again high (103.8°, pulse 132). There had been no chills. There was some sensitiveness to pressure over the tip of the mastoid. A careful general examination had been made without showing any other cause for the high temperature. That night, I opened the mastoid process. Pus was found just under cortex in small amount; there was softened bone leading backwards toward the knee of the sinus, but no pus,—no extradural abscess. The tip of the mastoid was free from pus. The wound was thoroughly cleaned and sutured. (The pus contained staphylococci). While the patient was under the anesthetic, a lumbar puncture was made. The fluid was clear and cultures were negative.

On the following day, February 18th, the patient appeared brighter and complained of no pain. The temperature had fallen to 97.8° for a very short time after the operation, but had risen again to 105° (in the axilla) at four-thirty that afternoon, (pulse 132). It steadily fell to 99.6° at midnight, (pulse 96), then again rose and on the following afternoon (February 19th) at four, reached 104°, (pulse 132). This was followed by another steady decline to 99° at four A. M., (pulse 88), on February 20th. The temperature remained low until midday, when it was 100°, but then there was a rapid rise to 103.4° at two P. M., 105.2° at four P. M. and 105.4° (rectum) at six P. M., (pulse 124). During these days, her general condition remained unchanged. She did not complain of pain; repeated examinations of her lungs revealed no signs of pneumonia. There was no indication of trouble in the abdomen which was carefully examined. There were no signs of typhoid, nor any symptoms of meningitis. The fundi of both eyes were normal. Calomel and salts had been given with effect. In spite of the absence of pain, of chills, of sweats and of any marked sensitiveness along the course of the jugular, I determined, against the opinion of two able colleagues, one a skillful surgeon, the other a well-known otologist, to

wait no longer, but to open the lateral sinus. The operation was performed that night, (February 20, 10 P. M.). The internal jugular vein was tied at a point about half way between the base of the skull and the clavicle; it was found collapsed; the wound was closed. The wound over the mastoid was opened and the sinus was exposed; it showed no marked change on the exterior; towards both ends it was bluish, in the middle whitish and slightly roughened, but a large thrombus was found within embracing the entire sinus down to the bulb and extending about one inch behind the knee, so that there was some bleeding at the lower end and a free gush of blood from the posterior. The thrombus was chiefly a solid white mass, but there was also some soft, broken-down pus. It was completely evacuated. The outer wall of the sinus was removed. Cultures and smears from the thrombus yielded staphylococci. The wound was packed.

During the next twenty-four hours (February 21st) the temperature did not go over 102° and the patient rested comfortably. But early on the morning of February 22nd, the patient complained of great pain in the right ankle and in the neck just below the wound. The temperature again rose, reaching 104.2° at ten A. M. and 104.8° at seven P. M., (pulse 124). The wound was dressed at midday; there was some swelling of the face and of the neck which was very sensitive to the touch. The ankle was very painful on pressure or when moved, but there was no redness nor swelling. The wound was dressed daily, the bulb of the jugular was washed out by means of a long, bent silver canula. But the temperature remained high, fluctuating between 102° and 104° for days. In the meantime, the right ankle became edematous and red and the swelling of the face disappeared, excepting in an area over the upper part of the jugular. On February 27th, fluctuation could be made out in the ankle. Leucocyte count 9,600.

February 28th. Deep punctures in the ankle gave no pus.

March 3d. A soft blowing systolic murmur was dicovered over the mitral area (which persisted and was present on May 1st, when patient was discharged).

March 4th. Under local anesthesia (ethyl chloride), an incision over the outer side of the ankle emptied two ounces of pus (smears and cultures from pus contained staphylococci).

March 6th. The scar of the jugular wound has opened and emptied of a small amount of pus, relieving the induration which

has existed since the operation.* The condition of the ankle has gotten worse; there is great swelling and much redness. Dr. Albertus Cotton was called in and under ether anesthesia, opened the ankle joint by a number of incisions. The joint was drained anteriorly and posteriorly with rubber tube drains, the other incisions with gauze. (The drains were all removed on March 10th). The patient, much debilitated by the previous suffering and fever, was greatly shocked by this operation but responded to stimulants.

March 14th. Patient complained of pain in the right hip. No redness nor swelling.. This gradually increased during the next two weeks. On April 3rd, there was considerable swelling over the hip and Dr. Cotton made a long incision under local anesthesia (cocaine) and evacuated about six ounces of pus. (Smears and cultures of the pus contained both staphylococci and streptococci.) A drain was kept in the wound for about ten days. Her temperature did not become normal and remain approximately normal until about the 22nd of March. By the end of April, her wounds had so far progressed that she was sent to her home on May 1st, where, since then, I am informed, she has completely recovered.

The case just described is one of great severity. The very high temperatures, the rapid prostration of the patient, the early occurrence of a metastatic abscess in the ankle and later in the hip make it evident that but for an early operation, the patient would not have recovered. The mastoid operation was performed twelve hours after the first high rise of temperature, the sinus was opened three days later, and yet the only sign upon which we could depend was the temperature. We must not allow absence of chills and sweats and pain to make the diagnosis doubtful, if the fluctuations of temperature and the height of the fever, which are characteristic, are present. Every other possible cause of the fever must be sought for as was done in this case, but delay is dangerous, and it is unnecessary and wrong to wait for more indications of the disease. In the next case, the operation was done still more promptly.

Case 3. W. T., a boy aged fourteen, was admitted into the Baltimore Eye, Ear and Throat Charity Hospital, September 6th, 1907, having been sent by Dr. Gambrill, of Alverton, Md. There had been no ear trouble until about ten days before admission, when he was kicked in the ear while swimming. There was some pain at the time which, however, ceased and the patient thought no more about

(2) Koerner recommends that the wound for the ligation of the jugular be left open at least above because of the tendency of the upper end of the jugular to suppurate. This case as well as the two following do not support this advice. See Monograph, 3rd ed., p. 131.

it until a day or so later, when a profuse discharge set in from the right ear together with febrile symptoms. Dr. Gambrill was called in and ordered irrigations. The discharge, however, continued profuse in quantity and the boy's general condition became worse. There was high fever, general depression and vomiting.

On admission to the hospital, examination of the right ear showed profuse discharge, which, on being cleaned away, rapidly reformed. Tenderness over the mastoid was especially marked over the tip and in the neck below the tip. Temperature 101°. Irrigations were ordered at once, but the boy's temperature steadily rose during the afternoon and evening until at 7 P. M. it was 105°, pulse 130. It was then decided to operate at once. Ether was administered. The usual curved skin and periosteal incision was made behind the right ear. On the first blow of the hammer over the antrum, pus oozed from the bone. The antrum was opened and found to be large in size and filled with pus and granulations. The tip cells were next opened. The mastoid was of the pneumatic type being honey-combed with small cells each filled with pus. When all portions of the mastoid seemed cleared, some new cell would be discovered. The process of cleaning away the cells was continued until there remained but a shell of bone. The sinus was not exposed nor was the dura at any point. The wound was irrigated with normal salt solution and the skin was closed by a subcutaneous silver wire suture.

September 7th. Patient fairly comfortable. Profuse discharge from the ear which was mopped out. Complains of some pain in neck below tip and this region is slightly swollen.

September 8th. Fairly good day. Temperature 100°.

September 9th and 10th. Complains of pain in neck and temperature remains rather constant between 99° and 99.5°.

September 11th. Dressing. Wound found filled with pus. Suture removed and wound broken down and thoroughly irrigated. Temperature began to rise at ten A. M. to 102.2°; at noon 104.5°. During the next fourteen hours, it varied between 104.8°, (pulse 120), and 103°, (pulse 100), then fell to 101.4°, (pulse 86), at eight A. M. on September 12th.

September 12th. Second dressing. Wound seemed in good condition. Less discharge from ear which up to this time has been most profuse. Temperature continues to rise high. At five P. M., temperature 106°, remaining over 105.5° from four P. M. till seven, (pulse 110). At seven, he had a "chilly feeling." Patient complains

of no pain. There is some swelling in the neck below tip of the mastoid which is tender. There has been no chill and no sweating. Thoracic and abdominal complications as well as erysipelas excluded. Bowels moved freely after calomel. Fundi of both eyes normal. Mental condition good, excluding meningeal complications. There being no possibility of retention of pus in the bone after so thorough an opening of the mastoid,—the diagnosis of thrombosis of the lateral sinus was made because of high fever alone. Under these conditions, it was decided to operate immediately and to ligate the internal jugular vein before opening the lateral sinus. The jugular was exposed at the point of election. The vein was rather small but not collapsed, though sometimes it would almost collapse while under observation. A single ligature was used to tie off the vein and the wound was closed with sub-cutaneous silver wire suture.

The mastoid region was next exposed; some swelling being noticed below the tip of the mastoid, in the neck the original mastoid wound was enlarged downward in a search for pus. None was found. The inner plate of the mastoid was then cut away until the lateral sinus was exposed. The bone wound was enlarged until the bone had been removed back beyond knee about one-half an inch and downward until the entire tip had been taken away. The lateral sinus showed no pus on the outer surface, but it seemed as if the lower portion was whiter than the portion above and perhaps flatter. At no point was there direct extension of the bone disease to the sinus though about the knee the diseased portion approached very closely. A fine incision was made into the sinus when a little oozing occurred, but no free bleeding. The wound was then enlarged and the thrombus removed. About one and one-half inches of the sinus wall was opened and removed. A little bleeding occurred at the lower end, free bleeding at the upper. The sinus was then plugged above and below, and a sterile dressing applied, after the mastoid incision had been sutured in its lower portion. Patient returned to bed in a very good condition.

September 13th. Patient does not complain of pain. Highest temperature 104.6°, pulse 100.

September 15th. No pain or discomfort. Highest temperature 103.4°.

September 17th. First dressing. Stitch removed from the neck, —wound had healed by first intention. Mastoid wound clean. No

bleeding from the sinus; wound repacked. Collodion dressing on the neck. Boy in excellent condition. Highest temperature 101.6°.

September 20th. Temperature rose to 102°.

September 21st. Second dressing. Wound looks well. General condition of the boy good. From this time on temperature normal.

October 3rd. Patient discharged to return for dressings. Wound granulating well. Sinus closed. Wound in neck perfectly healed. No swelling or pain anywhere. Patient last seen in the first week of November. The wound had then closed nicely by granulation and the boy was in excellent condition.

In this case it will be seen that conditions were similar to those in the preceding case. We had no sign as a guide except the fever. But here action was still more prompt. The temperature rose to 105° before the first operation, but after the mastoid had been opened, it fell steadily for four days. On the afternoon of the fifth day, there was a sharp rise followed by a slow fall, and in spite of dressings of the wound, a still higher rise of temperature on the following afternoon. The sinus operation was then done within thirty-six hours after the rise of temperature. The subsequent course was most satisfactory and almost the same as though it had been only a simple but extensive mastoid operation. The patient was discharged from the hospital in three weeks and in four weeks more the wound had closed completely.

It is safe to infer that it is to the promptness of the operation that the very favorable outcome is due. This case, even more than the former, bears evidence that the prognosis is the more favorable, the earlier the condition is relieved. But cases of early operation are not the only ones which have a favorable termination, as is shown by the following.

Case 4. Miss M. S., aged twenty-five, was admitted to the City Hospital on September 22nd, 1907. The patient taken ill one week ago to-day (September 15th). The illness began with aching in her left ear which had been discharging for the last four or five years. The discharge has been purulent, containing blood at times and had a very bad odor. Last week she had pain in that ear, and throbbing pains in her head. She has not been able to hear with that ear since the first attack. The discharge has now diminished. Has been vomiting during the last week. Bowels very loose. Loss of appetite. Not able to sleep at night on account of the pain. Some pain across the bridge of the nose. Has had chills with marked sweats.

Physical examination:—Face has a very anxious expression; eyes very bright; has a sallow complexion; tongue furred and heavily

coated. Extreme pain and tenderness to pressure over the region of the mastoid on left side. Temperature 104.2° this noon. Right ear normal.

Abdomen:—Very tender in the right iliac fossa, also over the region of her spleen and liver. Blood count, 10,400.

September 25th, 1907. Pain in head and at sterno-clavicular junction, also in knees. Herpes in both nostrils. Seems somewhat delirious at times. Widal negative. Blood examination for malaria, negative. White blood count 23,831.

I first saw the patient on September 26th. Her severe chills had been recurring frequently for twelve days; while at the hospital her temperature had frequently reached 103.5°, 104°, or 104.5° with drops to between 98° and 100°. Her sweats had been marked. There was no hard cord to be felt in the line of the jugular. The diagnosis was clear. I advised that the operation be done forthwith. The jugular was ligated first. Then the usual mastoid operation was performed. The bone was found highly sclerosed. Toward the antrum, broken-down cholesteatomatous matter was found. As the bone wound was enlarged in the direction of the lateral sinus, a cavity was opened out of which a large amount of pus evidently under marked pressure was emptied and continued to escape during the further progress of the operation. The antrum was found carious, but in view of the patient's weak condition it was deemed wise not to subject the patient to the radical operation at this time. The opening in the posterior part of the wound from which the pus was escaping was enlarged—and proved to be the sigmoid sinus whose outer wall had been softened and broken down so that the pus was found coming directly out of the sinus. The sinus was exposed in both directions and firm clots found toward the bulb and at some distance behind the knee. There was free hemorrhage when these were removed. Smears made from the pus coming from the sinus showed the organism to be the streptococcus.

The wound was packed and drained frequently. There was uninterrupted recovery. No chills occurred after the operation and the temperature rarely rose above 100°. On the second day, it rose to 101.8°, on the third to 101°, and on the twelfth day there was a sharp rise to 103.6°. But this dropped after a dressing of the wound and from this time on the temperature was normal. The patient left the hospital on the twenty-third day, in excellent condition; the wound healed entirely in about two and a half months. The otorrhea still persists.

The blood count in this case was interesting. At the time of admission (September 22nd) there was a leucocytosis of 10,400.

On Sept. 25th	23,800
On Sept. 28th, day after the operation	43,600
On Sept. 29th	21,400
On Sept. 30th,	12,400
On October 1st	14,000
On October 2nd,	13,700

The feature of special interest in this case lies in the explanation it offers of the favorable course and the absence of metastatic abscesses when chills and fever had existed for almost two weeks. The great central portion of the thrombus had become purulent but at both ends this purulent mass was closed by a firm clot, which kept the pus from entering the general circulation in spite of the high pressure under which it was held. To the success of this effort of nature, we must attribute many recoveries after operations long delayed. Reflection upon these cases convinces me that they also in a measure explain the varying attitude of operators toward the subject of ligation of the jugular vein. When a thrombus is so firm that it can resist the high pressure just described, and thus prevent the formation of metastatic abscesses, even after many days, it is probably as unnecessary to tie the jugular as it is essential to tie it in cases like number two and three. In the former it is probably inadvisable to break down the firm clot.

The views just expressed find corroboration in a case of what may be termed spontaneous recovery from sinus thrombosis which came under my observation some years ago.

Case 5. In December, 1903, I was called to see a child two years of age, who had within a few months suffered from an attack of scarlet fever, followed by measles. Double purulent otitis media was the result. A swelling had appeared over the left mastoid which a physician had incised three weeks before I saw the patient. I found a marked hard swelling about an inch behind the incision over the mastoid. The ears were both discharging. On the following day, the mastoid operation was performed. No visible opening into the mastoid was found in the surface of the bone which contained pus and granulations. The mastoid process having been cleaned out, a second incision was made one inch posteriorly into the abscess and much pus evacuated. The bone was found denuded. The patient made a good recovery but both wounds continued to

suppurate and excessive granulations formed. On January 20th, I dressed the wounds and found that a probe could be passed far into the posterior one, but it was not possible to determine its exact position.

On February 3rd, the patient having been admitted into the City Hospital, I found that the probe passed far into the skull cavity. An incision was made which joined the two previous incisions and the bone exposed. It was then found that a fistula extended from the posterior wound into the skull, the opening being situated in the masto-occipital suture. The opening was not more than five or six mm. in diameter and perfectly circular. It was enlarged and the bone removed forwards towards the ear, thus exposing the fistulous canal. I then found that the pus had not come from an extradural abscess, but that it was the lateral sinus itself which was continuous with the fistula. A grooved director was passed into the canal and it was slit open. In the sigmoid portion, the part was found where the sinus had been exposed to the mastoid disease and where the infection had occurred. It was evident that a septic thrombus had formed but had been securely shut off from the circulation by firm clots and that the pus had evacuated itself through the emissary vein, thus producing the abscess. The patient recovered nicely from the operation and is still under occasional observation for chronic purulent otitis media.

The case just described furnished an extreme example of the limitation of the septic thrombus which is to be regarded as a conservative effort of nature and which to a lesser degree is not of infrequent occurrence.

The cases just reported appear to emphasize:

First:—That the favorable prognosis of cases of thrombosis of the lateral sinus depends upon the surgical removal of the infected thrombus at the earliest possible moment, before metastatic infection has occurred or upon the effective limitation of the infected mass by a solid non-infected thrombus at both ends, and

Secondly:—That the early diagnosis may and should be made in those cases in which chills, sweats and pain are absent on the characteristic sharp rises of temperature to 104° and higher and the irregular fever, when other conditions which may cause high temperature can be excluded.

CANDIDATE'S THESIS.

THE TONSILLAR RING AND THE PARANASAL SINUSES AS PORTALS OF ENTRY OF INFECTIOUS DISEASES.

SYLVAN ROSENHEIM, A. B., M. D.

The cases to be here reported are deemed of sufficient importance and interest to justify this paper. I consider them an important link in the chain of evidence proving these parts to be frequent portals and foci of infection.

By the tonsillar ring we understand the faucial, lingual and pharyngeal tonsils, as well as numerous isolated and combined lymphoid follicles found in the oral, naso- and laryngo-pharynx.

The various views regarding the functions of the tonsils can be conveniently divided into two groups; first, those theories advanced before Stohr's discovery of the emigration of the lymphocytes from the interior to the periphery of the tonsils, and those advanced after the discovery.

The earlier writers, mostly anatomists, considered the tonsils as functionless organs, supporting in nature (Kolliker).

Morrell Mackenzie mentions a theory very commonly held at that time, of the relationship to the sexual organs, based on the frequently found hypertrophy at the age of puberty.

The earlier clinicians considered them to be of importance to the digestive functions. Bosworth considered that they acted mechanically in moistening the bolus of food. Rossbach thought they produced a saccharine ferment. Fox thought the faucial tonsils absorbed superfluous salivary secretion, and Spicer assumed that the pharyngeal tonsils did the same for the tears and nasal secretion.

That the tonsils are blood-forming organs has been suggested from time to time. Wood has renewed this theory with great

gusto in a recent article (1904). The discovery of the germinal centers, i. e., places in the follicles in which active karyokinesis and cell division is taking place, first gave rise to this view. To substantiate it, experimenters have determined a decrease in the number of lymphocytes in the circulation after extirpation of the tonsils. Pluder has described a constant emigration of newly formed cells into the efferent lymph channels, but Goerke has pointed out how impossible it is to demonstrate this. Goerke (1907) says that the tonsils as a part of the lymphatic apparatus may take some part in their blood-forming work, but by no means an important role. In a recent work (1904) Wood gives his revolutionary conclusion in regard to the functions of the tonsils. He considers them the principal leucocyte formers. They give rise to the lymphocytes, which in turn are transformed into the various other forms of leucocytes. They arise, he says, not from the germinal centers, but directly from the tonsillar crypts.

It has been thought that the tonsils formed an internal secretion, such as the adrenal bodies and thyroid produced. The experiments along this line have given opposite results. Masini injected an extract of tonsils into various animals, and ascertained a rise of blood pressure. Scheirer, in performing similar experiments, obtained a fall in the blood pressure.

In 1884 Stohr described a phenomenon that occurs constantly in the tonsillar tissue, namely, a wandering of the lymphocytes from the interior of the tonsils to the periphery. They force themselves or are forced between the epithelial cells, injuring them and destroying their function. The open spaces thus made between the epithelial cells led Gerhard to consider the tonsils as physiological wounds. Some consider the tonsils to be rendered much more vulnerable to deleterious substances and micro-organisms which might readily penetrate the tonsils, whereas others pointed out the fact that the constant streaming out of the leucocytes was enough to prevent this. The two opposing theories have been termed the protective and the infection theories.

To determine whether foreign bodies and micro-organisms penetrate the tonsils, Hodenpyl, Goodale, Hendelsohn, Lexer, Goerke and others have performed numerous experiments. Hodenpyl (1891) smeared human tonsils with olive oil and lard and applied carmine and berlin blue in a similar manner in dogs, from fifteen minutes to one-half hour. He also used solutions of analin colors

and atropin. He concluded from his experiments that they absorbed neither fluid nor solids under ordinary circumstances. He thought that the rarefaction of the tonsillar epithelium explained the entrance of the toxin of diphtheria into the general circulation.

Goodale (1898) and Hendelsohn (1899) arrived at different conclusions from their experiments. Goodale's experiments were performed on the human hypertrophied tonsils and seem most convincing. He injected aqueous carmine solution into the tonsillar crypts and then excised the tonsils at intervals of from twenty minutes to two hours. In two control cases he excised the tonsils immediately after injection, and these proved negative. In all the others he found absorption of the carmine particles, the depth at which they were found varied directly, according to the interval which had elapsed since the injection. Hendelsohn arrived at similar results by blowing the colored particles over the tonsils.

Goerke (1907) claims to have arrived at negative results in repeating these experiments, and ascribes the positive results to two factors; first the mechanical force used in doing the experiments, and secondly, to a reversal of the lymph stream due to the amount of powder used. He thinks that bacteria penetrate more readily the mucous membrane of the pharynx. He mentions some experiments of Lexer in which virulent bacterial cultures were painted over the throats of animals, and very few were found to penetrate the tonsils. This author, however, considered the lymphoid tissue of the throat to be the portals of entry. He also mentions some cases reported by Menzer of acute articular rheumatism, following angina, in which bacteria were found deep in the pharyngeal mucous membrane, and but few in the tonsils, and these superficial. He thinks the tonsils owe their protective function not only to the mechanical influence of the constant stream of leucocytes towards the surface, but to two other factors, namely, the bactericidal action of the lymph and of the products set free by the breaking down of the lymphocytes. That infections occur through the tonsils, Goerke doesn't deny; that they do not occur more frequently is due to their protective function. He says they reach their greatest development in childhood at the time when the infectious diseases are most common, and there is most need for them and they retrograde after this.

Whatever may be the true function or functions of the tonsils, we are inclined to agree from our studies and the results of a care

ful reading of the literature, with Bosworth, that normal tonsils should not be seen. Goodale's experiments were done on hypertrophied tonsils, and certainly showed that such diseased tonsils absorbed foreign materials. The evidence of the literature is also overwhelmingly in favor of this stand. As we shall see, many of the tonsils in severe septic infections appeared normal on the surface, whereas below the surface an entirely different picture was seen. We would not, however, restrict the diseased tonsils to those which project beyond the faucial pillars in case of the faucial tonsils, as we all know that some of the most badly diseased tonsils are those which are hidden. The history of repeated attacks of sore throat sometimes calls our attention to them in such instances, or we discover their condition in searching for a focus in some so-called cryptogenic infection. I think that everyone is agreed that any pharyngeal lymphoid tissue that is seen should be removed. This is subject to all the diseases that affect the faucial tonsils, and they probably are much more frequently inflamed than is supposed, but owing to their position this is overlooked.

As most of our tonsil cases had acute or chronic arthritis as sequelae, I shall first briefly review the literature of the other infectious diseases associated with diseases of the tonsils, before taking up the rheumatic cases.

Aneurysm. Libman reports a case of mycotic aneurysm which he ascribed to a tonsillitis which occurred three weeks previous to its occurrence. Death resulted after operation, and at autopsy streptococci and staphylococci were grown from the aneurysmal sac.

Appendicitis. Cases reported by Apolant and Kretz.

Erysipelas. Gerhard, focal erysipelas frequently has its origin in the naso-pharynx.

Meningitis. Perigord reports a case of fatal meningitis, in which at autopsy an abscess was found in the pharyngeal tonsil.

Iritis. A case in which this complication occurred is reported by Dock.

Pleuritis and Pericarditis. Numerous reports by Hanot, Heddaeus, Dock, Jessen, Richardieu, Lermoyez Metzner and others. In a case of Hanot's, streptococci found in both the tonsillar and pleural exudates. In Metzner's case the tonsils at autopsy showed collections of pus in the interior with the surface intact. Staphylococci and streptococci found here as in the pus in the mediastinum, pleural cavity and endocardium.

Pneumonia. In a young girl seen by Jessen an angina was followed in twelve days by pneumonia, pericarditis and nephritis. Streptococci found in the tonsils. Blood culture was negative.

Paraplegia and strabismus have been observed to follow tonsillitis by Emil Mayer, and parotitis by Rosenberg.

Nephritis. Leyden, Jeppel, Morse and others have laid stress on this complication of tonsillitis. Leyden and Morse call attention to the fact that some of these cases may develop into the chronic forms of nephritis.

Osteomyelitis. Kraske and Mayer report cases.

Trumbull reports a case of phlegmon of the lower extremities, following follicular tonsillitis.

Oophoritis and Orchitis. Joal reports six cases of this complication.

Septic Infection. Fraenkel, Heubner and Bahrdt, Jessen and Babes relate some interesting reports. Fraenkel describes a case of retro-pharyngeal phlegmon, which began in the tonsil, followed by purulent pericarditis and pleurisy. Micrococci were found in the exudate on the tonsils and elsewhere. In another case of membranous disease of the pharynx and larynx, ulcerative endocarditis followed. Diplococci and streptococci found. Heubner and Bahrdt had a case of fatal scarlatina and acute articular rheumatism, following membranous tonsillitis. The autopsy demonstrated a purulent exudate in the tonsil, which had penetrated the capsule, causing an infectious thrombo-phlebitis of the common jugular vein. Cocci in chains were found in the exudate on the tonsils, in the joints, blood, etc. Jessen reports two interesting cases. In one a woman was brought into the hospital comatose. There were hemorrhages into the skin. Albumin and casts were found in the urine. The tonsils and mouth were negative. Death occurred in twelve hours. Autopsy revealed abscesses in the interior of the tonsil on section, the outer surface appearing normal. Large soft spleen. Numerous abscesses and hemorrhages in the kidneys. In the second case the disease began as a follicular tonsillitis, which was followed by double pneumonia, pericarditis and sepsis. On sectioning the tonsils abscesses found, the surface being intact. Staphylococci found in the organs.

Skin Manifestations. Erythema, erythema nodosum, purpura, and erythema exudativum multiforme described by Jessen, Stephanides, Joal, Finger and others. In a case seen by Jessen, erythema

nodosum and pains in the joints followed three days after the onset of an acute follicular tonsillitis. Streptococci and staphylococci were found in the tonsillar exudate in pure culture. Blood culture was negative. In the case of a woman subject to attacks of purpura and polyarthritis seen by Stephanides, removal of the tonsils resulted in a cure. Finger found streptococci in the skin of a patient with erythema exudativum multiforme.

Capalarri reports five cases of typhoid, with an angina at the onset.

Tuberculosis. The frequency of tuberculosis of the tonsillar ring is now so generally recognized that it is rare to see an article, as that of R. C. Brown, in which it is declared to be infrequent, as an argument in favor of the protective function of the tonsils. Meyer originally expressed the idea in a vague way that enlargement of the lymphoid tissues in the throat might in some way be related to scrofula. Lartigau and Nicoll in an article written in 1904 have given an extensive review of the subject, together with the result of their examinations of seventy-five pharyngeal tonsils. Parts of each tonsil were used for inoculation experiments, the rest were examined histologically. They found that 16 per cent were tuberculous and 10 per cent showed histological lesions of tuberculosis. The lesions are usually close to the surface and focal in character. They considered that the tonsils might be portals of entry for tuberculosis. From a careful histological examination of sixty-four adenoids, Uffenorfe found that 4 to 5 per cent were tuberculous. Two of these cases gave no tuberculin reaction after their removal; in one in which the patient had also pulmonary tuberculosis, there was a reaction. The author concluded from this that the lesions were purely focal. Moritz Schmidt says it is remarkable how infrequent there is tuberculosis of the tonsils when one sees how frequently it is seen post-mortem (Strassman 8 per cent, Dmochowsky 39 per cent, Schlenker 8 per cent). However, the later researches, as in that of Lartigau and Nicoll, have shown a high percentage of tuberculosis in adenoids removed during life. Often numerous specimens have to be examined before this is demonstrated. The tubercle bacillus penetrates the tonsillar epithelium in the lower animals without leaving any traces (Theobald Smith), and there is no reason why the same should not occur similarly in human diseased tonsils. In a number of cases of cervical adenitis, in which the tonsils on removal were given a care-

ful examination, both histologically and by inoculation of animals, Goodale found a large number to be tuberculous. The prompt clearing up of the glandular condition in these cases demonstrated the primary nature of the lesion in the tonsils.

The literature showing the relationship between the various forms of rheumatism and tonsillitis is very extensive. The connection between acute articular rheumatism and angina has been long noted. Heberden in 1804 described an angina which terminated in acute articular rheumatism, and Trousseau in 1865 revived this idea. Friedrich points out that it was one of the first diseases in which the tonsils were recognized as a portal of entry for infectious diseases. The importance of angina as an etiological factor was also early pointed out by Lagraniere, Boeck, Loebl, Mantle, Lennox Brown, Fowler, Cheadle and others. The literature of the subject is completely covered in the articles of Buss, Suchannek, Bloch and Peltersohn.

St. Clair Thomson says that 30 per cent. of the cases of acute articular rheumatism are preceded by angina. According to Ingals 13 to 29 per cent of cases of acute articular rheumatism and tonsillitis have an identical cause.

Stoffel, Stephanides and Gurich have reported cases of cures of the arthritis following proper treatment of the tonsils, and the latter makes this a point in favor of the tonsil being the portal of entry.

A great variety of organisms, staphylococci, streptococci, pneumococci, etc., have been found in angina. Likewise various organisms have been found in acute articular rheumatism. Poynton and Paine in eight cases found a diplococcus which they demonstrated in the tonsils, the pericardium, the heart valves, and the rheumatic nodule. They review the literature and state that the following organisms have been obtained: Staphylococci and streptococci, diplococci and anaerobic bacilli resembling anthrax.

I shall first briefly describe the technique and findings in a small series of cases in which I removed the tonsils, which were studied bacteriologically by Dr. J. C. Meakins. Most of these were from the wards of the Johns Hopkins Hospital, and represent the cases of acute articular rheumatism, admitted there over a short period.

All these were cases in which the faucial tonsils were at fault, and concerned adults. They were enucleated under cocaine anesthesia by the method described by Worthington. They were car-

ried in sterile receptacles to the laboratory and cultures taken in three ways, which Dr. Meakins calls external cultures, internal cultures and autolized cultures. The external cultures were taken from the depths of the crypts. Cultures from the interior were taken through a sterilized cut surface from the interior on slant glycerine agar and agar plates. The autolized cultures were made as follows: The tonsils were first washed for one minute in a 1-1000 solution of bichloride of mercury, then in sterile salt solution, after which they were placed in either broth or milk culture flasks.

External Cultures. In all of these streptococci and staphylococci predominated. Other organisms also found, either bacilli of diplococci.

Internal Cultures. In five cases the streptococcus was found in pure culture. In four streptococci and a few staphylococci found. In one streptococci and a few bacilli and in one negative results.

Autolized Cultures. In seven pure culture of streptococcus. In one streptococci and a few other cocci (4 foreign colonies in two plates). In one the staphylococcus pyogenes aureus principally. In two the results were negative.

Case 1. Mrs. C. G., aet. 40 years, white. Admitted March 18, 1907. Discharged May 1, 1907. Diagnosis: Acute articular rheumatism. Complaint: Sore throat and rheumatism.

F. H. Negative.

P. H. Usual diseases of childhood, but no diphtheria or scarlatina. Has suffered from tonsillitis from her 13th year. When 14 years old had an attack of acute articular rheumatism affecting both ankles and wrists.

P. I. Began seven weeks ago with an attack of quinsy in which the abscess ruptures into the throat. Two weeks ago both shoulders, hips, knees and elbows became very tender, painful, hot and slightly swollen.

P. E. Joints above mentioned painful and tender, but not swollen. Slight mitral systolic murmur.

Very little tonsillar tissue seen. Slight amount of exudate in the crypts. Cultures from the throat gave almost pure culture of streptococcus. Opsonic index to streptococcus 0.65.

The tonsils on dissection proved to be much larger than expected, belonged to the almost completely submerged variety.

May 1. Patient left hospital much improved, still having some pain at times and slight stiffness.

The internal and autolized tonsils showed pure cultures of streptococcus.

Case 2. Mr. A. M., aet. 19 years, white. Admitted March 20, 1907. Discharged April 26, 1907. Diagnosis: Acute tonsillitis. Acute articular rheumatism. Acute mitral endocarditis. Ascaris and trichiuris infection.

F. H. and P. H. Unable to obtain.

P. E. Joints affected. Pain in right elbow, hips, right knee and behind neck. Elbow is hot, red and swollen. The tonsils on admission were large, red and acutely inflamed. No exudate present. On the post-pharyngeal wall is a thick, greenish, purulent secretion. Pus is seen in the right naris, about the middle turbinate and on the inferior turbinate. On exploration of the antrum about a week later a slight mucoid discharge found.

April 12. Tonsils dissected out.

April 26. Discharged. Still has at times slight pains in joints. Has improved very much and has gained weight.

Internal cultures showed pure streptococcus. Autolized cultures negative.

Case 3. Miss H. B., aet. 32, white. Admitted February 28, 1907. Discharged June 10, 1907. Diagnosis: Arthritis deformans. Acidosis. Complaint: Stiff and swollen joints.

F. H. Negative.

P. H. No history of tonsillitis. Has had an occasional sore throat. As a child had "catarrh."

P. I. Duration four years. Knuckles gradually enlarged, no pain. One year later the wrists affected, swollen but not painful. Two years ago shoulders. Now right knee is swollen and painful.

P. E. Nasal examination revealed no signs of accessory sinus disease. A few diseased crypts found at the apex of an atrophied tonsil.

A slight amount of tonsillar tissue removed by dissection. Cultures from this, internal and autolized demonstrated streptococcus alone.

She was treated with injections of dead streptococci.

When she left the hospital June 10 she was much improved.

Case 4. History lost.

Case 5. L. S., aet. 29, white. Admitted March 27, 1907. Discharged April 30, 1907. Diagnosis: Acute articular rheumatism (second admission).

F. H. Unimportant.

P. H. Negative. No history of tonsillitis or sore throat.

P. I. In 1903 pain in various joints, but no redness or swelling. Was unable to walk or move legs.

Complains of pain in left knee and sterno-clavicular joints. Was hoarse and had sore throat several days before this attack.

P. E. Left knee joint swollen, hot and red. Diffuse tenderness. Right ankle swollen.

A little muco-pus in left nostril about middle turbinate. Tonsils enlarged, otherwise not diseased.

April 19. Small fibrous tonsils dissected out. No rise of temperature following operation.

Discharged well April 30.

Internal cultures from tonsils showed streptococci and a few staphylococci. Autolized cultures contained pure streptococcus.

Case 6. History lost.

Case 7. Dr. A. D. H. Tonsils removed as a prophylactic measure after an attack of tonsillitis. Patient was subject to frequent attacks of tonsillitis. These, however, had not resulted in any of the sequelae. Both internal and autolized cultures showed pure streptococcus.

Case 8. Mr. W. F. P., aet. 26 years. Admitted April 26, '07. Discharged April 30, '07. Diagnosis: Hypertrophied tonsils. Acute arthritis (convalescence). Mitral stenosis. Complaint: Patient is convalescent from an attack of acute arthritis and comes into the hospital to have tonsils removed.

F. H. Unimportant.

P. H. Has had measles, mumps, pertussis, scarlatina and rheumatism. Had scarlatina when 14, and rheumatism (knee joints swollen and painful), when 16 years old.

P. I. Began March 8 with headache and slight sore throat. March 13, began to have severe cough and later expectorated blood. On March 15, knee joints became stiff and painful. These cleared up in a few days, when he began to have shortness of breath, and had pain in the chest, running down the arm.

P. E. Above diagnosis.

April 26. Dissected out large submerged tonsils. Considerable hemorrhage at night, necessitating the use of Monsel's solution Temperature for two days after raised to 100.5° and slight pain in ears.

Both internal and autolized cultures showed pure streptococcus

Case 9. Miss E. W., aet. 32, white. Admitted May 20, 1907. Discharged May 21, 1907. Diagnosis: Acute infectious arthritis Chronic tonsillitis. Complained of enlarged tonsils.

F. H. Unimportant.

P. H. Tonsils have been enlarged since childhood No history of attacks of tonsillitis or quinsy.

P. I. Five weeks ago had swelling of right hand and forearm, which was pronounced inflammatory rheumatism by her doctor Had pains in various parts of her body. This lasted two weeks.

P. E. Knuckles of first and second fingers are swollen. Hands are held flexed and cannot be completely extended. Exostosis on right wrist. Both ankles slightly enlarged, motion is not impaired. Tonsils on dissection proved to be very badly diseased The crypts were full of a cheesy looking material. Temperature 100° for one day after operation, then fell to normal.

Internal cultures in these tonsils demonstrated streptococci and a few bacilli. Autolized culture showed pure streptococcus.

Case 10. Mr. N. E. S., aet. 30 years, white. May 20, 1907. Diagnosis: Acute articular rheumatism. Hypertrophied tonsils. Complaint: Rheumatism.

F. H. Mother suffers at times from rheumatism. Otherwise unimportant.

P. H. Has had measles, diphtheria, scarlatina, mumps, chicken pox and typhoid fever.

P. I. In the latter part of February had an attack of tonsillitis This was immediately followed by pain in the left knee, and afterwards in numerous joints. Has just come from Atlantic City, where he has been during this illness.

P. E. The joints of the lower extremities are somewhat stiffened, though the function is fair. Slight thickening of the joints of the upper extremity. Tonsils hypertrophied.

May 20. Tonsils dissected out. Were much larger than apparent from the surface. No rise in temperature after operation

Urine negative.

December 12. Patient is in very good health. No signs of his rheumatic trouble (report from mother).

Internal cultures demonstrated mostly streptococci, and a few staphylococci. The autolized cultures were negative.

Case 11. Mr. B. S., aet. 27 years, white. Admitted April 27, '07. Discharged July 2, '07. Diagnosis: Acute articular rheumatism. Chronic tonsillitis.

F. H. Unimportant.

P. H. No history of attacks of tonsillitis. Had an acute coryza two weeks previous to this illness.

P. I. Began three weeks ago with pain and swelling in the left ankle. Then the other joints of upper and lower extremity became affected. On admission right elbow acutely inflamed. Both shoulder joints and left ankle very tender.

P. E. Joints affected as above described. Heart normal. Both tonsils very large. Pus seen in right naris, about the middle turbinate and over the inferior turbinate Pharynx and post-nasal space atrophic. Vocal cords reddened.

Tonsils removed May 23. May 31 a note made saying that arthritis has wonderfully improved.

July 2. Is now able to walk. Temperature at times goes up to 100°. No pain on passive motions of the joints excepting the left knee.

Internal cultures of these tonsils were negative. Autolized cultures showed streptococci with four colonies of a foreign coccus in two plates.

Resumé of cases and conclusions regarding tonsils.

In two of the series of cases which Dr. Meakins studied bacteriologically the histories have been lost. In one case the tonsils were removed as a prophylactic measure several weeks after an attack of tonsillitis. These were of the submerged variety and both internal and autolized cultures demonstrated streptococci in pure culture. Seven of the remaining eight were cases of acute articular rheumatism. In five of these, the tonsils were definitely hypertrophied, in two they were not. In four of them the streptococcus was found in pure culture, in two streptococci and staphylococci, in one streptococci and other cocci, in one streptococci and a few bacilli.

In one case of arthritis deformans streptococci were found in pure culture. This case was treated with streptococcus vaccine and improved markedly.

In five of the arthritis cases no note was made in the history of previous attacks of tonsillitis. At the onset of the disease in one case, the patient had "sore throat," another had quinsy, and another simple tonsillitis. One patient had two weeks previously an acute coryza. No note made, in the remainder, of throat symptoms.

Two of the cases had serious complications, an endocarditis of the mitral valves. Both of these were cases in which the streptococcus was found in pure culture internally. In one of these the opsonic index to the streptococcus was 0.65. In another case of acute articular rheumatism complicated by mitral and aortic disease, streptococci were found in pure culture in the tonsillar crypts and in this case the opsonic index was 0.55.

All of these cases did well, leaving the hospital a short time after the removal of the tonsils either well or much improved.

Besides this series of cases of acute arthritis, I have had a number of the more or less chronic variety, but sufficient time has not yet elapsed to draw conclusions. In all, however, the tonsils, even though not apparently diseased, have disclosed evidence of disease in their removal. In some pus was seen to exude from between the anterior pillar and the tonsil, in others cheesy matter was expressed from the crypts. Even though this may not be seen on removing them, careful bacteriological examination will often reveal virulent organisms in the depths.

Our conclusions from the above observations are:

(1) That bacteria penetrate deeply into the substance of diseased tonsils.

(2) That all tonsillar tissue that appears hypertrophied should be treated or removed. All evidence points to the ready penetration of bacteria and injurious substances into their interiors, in many cases leaving no visible sign of such penetration. In the present state of our knowledge we have no method of pointing out the dangerous varieties of tonsils. Any functions that they may perform will be carried on by the tonsillar tissue that always remains, even after the most careful removal. There are no reports in the literature of damage done by such removal.

(3) In all cases where there has been a history of repeated attacks of tonsillitis, this should constitute a cause for their removal, although they do not look diseased.

(4) In all infectious diseases the lymphoid tissue of the throat should be carefully examined as a possible focus of infection.

(5) The marked and immediate improvement in the series of cases reported, substantiates the above conclusions.

(6) The organisms found in the tonsils in the cases reported are probably the causal agents of the acute articular rheumatism attacks.

The Paranasal Sinuses as Portals of Entry.

I wish to report a case which strikingly illustrates this source of infection. We are just as much in the dark in regard to the functions of these cavities as we are regarding those of the tonsillar ring. Accumulating evidence seems to point to them as a frequent focus of distant infectious trouble.

We have a number of carefully worked out series of autopsies on these cavities by various authorities. Fraenkel found 43.1 per cent of diseased sinuses, Gradenigo 22 per cent (antra only examined), Lapalle 32 per cent, Martin 48 per cent, Kirkland 32 per cent, Wertheim 47 per cent, Oppikofer 48 per cent.

All agree in the great percentage of diseased cavities, but there is considerable difference of opinion regarding its significance. Frankkel, Werthein, Kirkland, Darling consider that it may be a source of general infection. Wertheim mentions three ways in which this occurs, (1) Swallowing the secretion, (2) Aspiration of the secretion into the lower air passage,. (3) Absorption of toxins. I would like to add to this a fourth, namely, absorption by the blood or lymph of the causal organism. As yet we must rely mainly on our clinical data to prove this, for as yet there are no more exact methods.

Kirkland examined the various cranial sinuses in 100 consecutive autopsies at the Sydney Hospital. He found a remarkably high percentage of the patients dying of pneumonia had suppurative disease of these cavities (50 per cent). He considers that the sinus disease is secondary to the pneumonia. He reports some interesting cases in which he considered the sinus trouble to be primary, cases of pneumonia and peritonitis, bronchiectasis, bronchitis and gangrene of the lungs. In two cases, bronchitis was cured after treating the antrum of Highmore.

In 360 nasal obductions Wertheim found 165 cases of diseased sinuses, i. e. 46 per cent. Of these 96, or 23 per cent, were cases of empyema. He also found a high percentage of pneumonia cases had sinusitis, nine out of sixteen cases, namely 50 per cent. He says that tuberculosis predisposes to sinus trouble. He found 31 empyemata in 106 cases of tuberculosis, 29 per cent. . All of four cases of syphilis had empyemata. The author notes the difficulty in determining whether the disease is acute or chronic, but thinks that the majority of his cases were of the chronic variety. He mentions the fact that even clinically in both acute and chronic sinus cases, on opening the sinus at operation secretion may be present in but small amount, or altogether absent. The usual findings in his cases were an infiltration of the epithelium, with numerous polymorphonuclear leucocytes, round cell infiltration in the subepithelial zone, less in the periosteal zone, and an edematous infiltration of the entire tissue. A sclerotic condition was rarely found.

In a recent communication, Darling gives the results of 52 autopsies in which the various sinuses were examined. Of there 37 were cases of pneumococcus infections, 15 were control cases. Of the pneumococcus infections 92 per cent had purulent sinusitis, of the controls (anemia, intestinal trouble, broncho-pneumonia, etc.), but 28 per cent had a sinusitis. Of the pneumococcus cases there were 22 cases of lobar pneumonia, 9 of acute meningitis, 1 case of acute pericarditis, and 5 of septicemia. This writer considered in all these cases of pneumococcus infection, that the changes in the sinuses indicated a chronic inflammation. He considers the accessory nasal sinuses as the portals of entry of the pneumococcus.

In a recent article (1906), Oppikofer gives his conclusions on this subject. These were based on the results of 200 nasal obductions. He found 47 per cent of the cases had inflammation of the sinuses, in about 25 per cent these were purulent. He says it is practically impossible to determine whether these are acute or chronic by the histological examination. He considers that most of these cases are of the nature of terminal infections. He says that in 23 of these cases careful rhinoscopic examination showed sinus disease in but three.

In the case we are about to report the relationship between the sinusitis and the joint trouble can hardly be disputed. It illustrates very clearly that we must be on the lookout in regard to

these cavities as a possible focus of infection. Our diagnostic methods are still imperfect, and there is no doubt that even the most expert rhinologist overlooks cases of this kind. The great disproportion between autopsy statistics and clinical facts points to this conclusion. We cannot agree with Oppikofer that the majority of these cases are terminal infections, first because in certain diseases, e. g. pneumonia, such a large number of cases occur, and secondly, the histological changes negative this view.

Mr. E. B., white, aet. 31 years. Carpenter. Diagnosis: Spondylitis. Chronic sphenoiditis and ethmoiditis (R). Complaint: Referred to me by Dr. Baer for examination of the nose and throat, as he could not lie on his back on account of secretion flowing back into the throat.

F. H. Unimportant.

P. H. Some years ago had suppuration in the right middle ear. Has had nasal trouble for years. In July, 1906, patient was treated in the Johns Hopkins Hospital for pain in the hips and in the back of the right leg. After a short rest in bed, he left the hospital much improved. In November, 1906, the pains returned. There was marked sciatica, and the spine was stiff and bent to the right. The patient was scarcely able to sleep at night.

P. I. In January, 1907, he was again admitted to the hospital with the same condition. He was unable to bend the spine. He has had a discharge from the nose for six years. He has pain in the back of his head and his ears at times.

P. E. An abundant purulent discharge is seen in the right naris between the middle turbinate and septum. The turbinates are atrophic. The left naris is negative. The pharynx and post-nasal space are atrophic. The posterior wall is covered with muco-pus. The larynx is normal.

Treatment: Up to this time the treatment of the spondylitis had been ineffective. He was unable to lie quietly on his back, and the bakings did not do much good.

The right middle turbinate was removed and the posterior ethmoidal cells and sphenoidal sinus were laid wide open by means of Hajek's hooks and the Cordes sphenoidal instrument. There was considerable necrosis of these cells, and they were thoroughly curetted. This operation was done in January. The improvement in the spondylitis was immediate. Dr. Baer said it cleared up as if by magic. In February the nose looked very clean. In March

he was shown at the Johns Hopkins Medical Society, when his back was straight and there was no limitation of motion.

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PAPER:

A BRIEF CONSIDERATION OF THE PROGNOSIS IN DEAFNESS DUE TO CHRONIC NON-SUPPURATIVE DISEASE OF THE MIDDLE EAR.

THOMAS JEFFERSON HARRIS, M. D.

The pendulum of thought in otology has, beyond question in recent years, been in the direction of surgery. No region of the body shows more brilliant advances in this direction in the last twenty years than the ear.

To those conversant with the subject, the amount of investigation and research from the surgical standpoint is stupendous, and to all, the fruits of these labors in life saved, is a subject for everlasting praise. What more alluring and inviting a quest than by skillful and able and timely interference to relieve the sufferer from the certain fate that otherwise awaits him. Far be it for any one to do aught but to acclaim with dying breath what the clinician and surgeon have together accomplished in this direction—medicine has no more brilliant page.

Surgery can, however, at the most fill but a small share of the average physician's life. Certainly it represents but a part, though a very important part of the science of otology. Much more commonplace, but no less important, viewed from the standpoint of usefulness to the community, are those chronic affections of the ear which lead to permanent, and in many instances, excessive impairment of the hearing.

The problem for relief is here a much more intricate one than in acute conditions demanding surgical intervention. To be frank, the promises of return are so discouraging that comparatively few have felt called to take it up. The ordinary meeting of our otological societies is given up to an account of operations and their results. A perusal of otologic literature will bear out the assertion that with the exception of William Meyer's thesis on Aden i ls-

and the subsequent unanimous agreement as to their baneful effects on the middle ear, little of value has been contributed since Toynbee's classical work.

Aside from the lack of incentive which acute diseases always call forth, it is to be freely conceded that the subject is a discouraging one—to many, a hopeless one. The region involved is almost microscopic, the opportunities for exact study in consequence exceedingly restricted. Yet in spite of all this thoughful minds have wrestled with the problem; for the files of our medical journals abound in remedies and original lines of treatment which though promising much, have for the most part unfortunately failed to stand the test of time. What then is the position of non-surgical otology to-day? What prognosis shall be attached to non-suppurative affections of the middle ear? In other words, what encouragement can be conscientiously offered to the sufferer from chronic middle ear deafness regarding restoration or improvement of hearing, provided he submit to the most approved form of treatment known to-day? As old as otology is itself, is the question, and yet as pertinent at the present moment as when first asked. All intelligent treatment must be based on a correct understanding of the pathologic condition of the affected parts. Empiricism may succeed elsewhere, scarcely in combating diseases of the ear.

Starting the consideration of the question under discussion from this standpoint, we are at once impressed by the small amount of pathologic investigation that has been made. All modern pathology of the ear hies back, and is dependent on the work of Toynbee, now fifty years old. While it is true that Politzer, Lucae, Siebenmann and others by their laborious efforts have amplified and perfected Toynbee's work, it still holds true in its main details to-day. Accepting this as the basis, writers on otology generally recognize two forms of chronic non-suppurative diseases of the middle ear; one, the *hypertrophic*, and the other, the *hyperplastic*, or *sclerotic*. It is important, however, to bear in mind that in many cases a sharp division into these classes is not possible. As Politzer well states, the hypertrophic and hyperplastic forms may proceed hand in hand. In any case, the final stage of the hypertrophic form is one of hyperplasia. Indeed while it is generally held that the by no means rare hyperplastic form known as otosclerosis develops without any previous hypertrophic stage, Lucae, in his recent book on "Die Chronische Progressive Schwerhörigkeit," asserts that in the

great majority of cases it proceeds from a previous catarrhal or suppurative middle ear disease. Another point in the pathology is of interest, namely, that there occurs a secretory stage, upon which much emphasis is placed by Politzer. In this country, at least, this form characterized by the presence of mucus or serum in the middle ear is seen but rarely. It is clear, however, that such a condition may exist at one time or another in the course of the disease. We must further remember, in considering the prognosis, that both the hypertrophic and hyperplastic form of the disease may come under observation at any stage from incipiency to its termination. A typical case of hypertrophic middle ear disease would then show at the beginning more or less hyperemia of the tympanic mucosa depending on the swelling and closure of the Eustachian tube. This advances next into a distinct edema and swelling, with accumulation of serum or mucus. This swelling can be confined to limited portions of the tympanum or involve the entire cavity. The secretion is discharged through the drum membrane or Eustachian tube, or is absorbed until finally in place of the simple hypertrophy, organized tissue appears. This occurs in the form of adhesive bands binding down the ossicles and involving the various ligaments and causing as a result, the characteristic changes in the position of the ossicular chain and the drum membrane. Such deposits of connective tissue will advance until the entire ossicular structure lying in relation to the inner walls of the tympanum is involved and anchyloses take place. In a case of pure oto-sclerosis, a similar gradual advance in the pathologic process takes place. Not immediately is there the complete anchylosis which is seen so often in the cadaver where the stapes and oval window are welded into one mass of bone. At first there is only a rigidity and suggestion of such anchylosis and later a broadening of the foot plate and only finally the excessive deposit of bone. This gradual progress in the pathologic state, both in the hypertrophic form and in oto-sclerosis must be constantly borne in mind. Too often an inexcusable degree of carelessness is seen in our modern text books in the treatment of this question of *prognosis*. With a sweeping assertion all cases are dismissed as curable or incurable without any consideration of the particular pathologic condition present, or the degree to which it has advanced.

With the same lack of proper consideration, the question of the *etiology* of these affections is treated. If it is true that no in-

telligent treatment can be undertaken without a knowledge of the pathologic process existing, it is doubly true that a clear appreciation of the causes producing it must be kept in mind. An inspection of the majority of the recent treatises on the ear will cause surprise at the hasty and imperfect way in which this question is handled. Most writers regard it sufficient to say that all non-suppurative diseases of the middle ear are dependent upon diseases of the nose and throat, though some of the older writers are not so sweeping in their assertions, Toynbee and Politzer, especially, placing emphasis on a number of causes more or less apparent. The relation of the nose and throat to diseases of the middle ear is without question an intimate one, and no one can intelligently deny the fact that in a certain, and probably a large number of cases, the cause of the ear trouble is to be found in either the nose or throat. That this is true in every instance is very much to be doubted, especially as regards the nose. Every otologist to-day admits the intimate relation that adenoid growths in the naso-pharynx bear to diseases of the ear. But the relation between the nose and the ear is by no means so certain. In a paper read before the Section on Laryngology of the New York Academy of Medicine a short time ago, the writer considered this question at some length and drew the following conclusions:

First. The nose plays an important role as a causative factor in many cases of otitis media, but by no means in all cases.

Second. The lesion in the nose is usually of an obstructive nature, acting as an obstacle to proper ventilation of the middle ear.

Third. In the beginning cases of hypertrophic otitis media a certain amount of improvement in the hearing can be confidently expected by restoring proper ventilation of that cavity through measures addressed to the nose, with the aim of relieving naso-pharyngeal and tubal inflammation; but (a) only such cases of the disease call for nasal treatment as show pathologic changes in the nose which themselves demand attention apart from the condition of the ear; (b) it is important to determine the true nature of the process in the middle ear, as the sclerotic or so called hyperplastic form is not influenced at all by such treatment; and (c) adhesive changes and anchyloses cannot be expected to yield, however completely the nasal obstruction is removed. In a word, while suitable cases are capable of being helped, many cases of chronic otitis media associated with certain nasal obstructions do not call for aid and will

not be improved by any form of nasal treatment, and all such treatment in these cases is unjustifiable and unwarranted.

Fourth. An important result to be secured by treatment is the relief afforded from the repeated attacks of acute rhinitis which by their effects on the Eustachian tube are wont to aggravate the chronic condition.

These conclusions represent on the part of the writer the conviction that many cases of middle ear deafness are not due to any cause lying within the nose. Since his attention has been called to the subject, he has been surprised at the number of such cases which present no gross lesions in the nose; the septa are comparatively straight, the turbinate bodies are not enlarged nor in contact with the septum. Particularly true is this in the cases of otosclerosis. Here not merely was there nothing strikingly pathologic to be discovered, but no history was obtainable of trouble at any time referable to the nose. We would not carry this contention as far as to maintain that gross lesions, such as marked deformities of the septa, polypi, etc., do not produce at times the affections of the ear in question, although patients are continually met with suffering from such lesions, without any symptoms referable to the ear. Obstructions in the posterior portions of the nose, however, are more accountable for such aural conditions and in particular the polyp-like hypertrophies of the inferior and middle turbinates. Just how these chronic forms of nasal diseases act upon the ear is not definitely settled.

In a recent discussion on this subject before the Section of Laryngology and Otology of the American Medical Association, one writer went so far as to claim that "this pernicious influence was the result of occlusion of the olfactory regions of the head and, as a result, deflection of the current of air into the lower part of the nostril and its escape by the Eustachian orifice." This assertion is certainly not borne out by the cases which the writer has seen and as has just been stated, it is the rule to find the middle meatus in such cases in a normal condition.

When we come to the throat, the situation is quite different. More and more, we are impressed with the evil effect on the ear of adenoid growths. In this respect the writer has been compelled to revise his opinion very decidedly. Formerly he held the view that in only such cases as showed a considerable amount of lymphatic tissue, could harm result to the ear. To-day he is of the

opinion that no rhinologist, however skillful, can say how small the amount is which may produce such results. Repeatedly do we meet with cases where the removal of the very smallest amount exerts a beneficial effect upon the ear. It must be further borne in mind that the natural history of this lymphatic tissue is to atrophy and disappear. It is on this account generally agreed that the cause of the ear trouble, if it is in the naso-pharynx, may no longer be ascertainable.

In just what way the adenoid tissue acts is also not fully demonstrated. Certainly not by mechanical obstruction as was formerly supposed. Presumably the inflammation is conveyed through the Eustachian tube by continuity or contiguity. In a similar manner, enlargement and disease of the faucial tonsil acts as a cause of middle ear disease. Indeed there are many who hold that the pressure of a large tonsil on the palatine muscles attached to the Eustachian orifice is a potent factor in producing such diseases.

The throat as we know is teeming with bacteria, many of a pathogenic form. Whatever lowers the defensive armor of the mucous membrane of the Eustachian tube, serves to allow them to enter the middle ear. Indeed it is fair to assume that there is no difference in the mode of invasion in the suppurative affections and in the so-called catarrhal processes save one of the virulence of the infection.

Back, however, of all these pathologic conditions in the throat and nose which are regarded as factors in producing the trouble in the ear, we would urge a common constitutional cause, and it is this cause which must be sought in every case which presents itself to us. We would not for a moment dispute the importance of establishing normal nasal respiration and of putting the nose and throat when called for, into the healthiest condition possible. This has of late been dwelt upon by Fitzgerald and with no more emphasis than its importance demands. It is no new theory, however—Cassels quoted by Roosa thirty years ago, urged the same views in a paper entitled, "Close your mouth and save your life." In this paper he refers to the remarkable hearing of the North American Indians and says that Catlin, a careful observer, never saw in his travels among them one who was deaf.

In arriving then at a just appreciation of the proper prognosis to be attached to chronic non-suppurative diseases of the middle ear, the following points have to be considered:

First. The form of the pathologic affection.

Second. The degree to which the process has extended, and specially whether the labyrinth is involved.

Third. The cause producing that process.

This, as will be seen, is quite at variance with the common practice of forming our prognosis merely according to the form of the affection, and of pronouncing all cases incurable where the disease has been existent for any length of time. Our prognosis will be governed further by:.

First, our ability to obtain proper ventilation where this is lacking in the tympanic cavity.

Second, our ability to maintain this ventilation, and,

Third, our abilty to absorb the pathologic products present.

I. Ventilation of the middle ear by opening the Eustachian tube by means of the Politzer bag, catheter or bougie has been a classic procedure for many years. Aurists are, however, more and more coming to the conclusion that this treatment has been too often undertaken in an irrational and ill advised manner, even in hypertrophic otitis, where it is alone justifiable. In spite of the views of certain writers to the effect that occasional inflation exerts a permanently beneficial influence, it is evident that such opening of the Eustachian tube and re-establishment of the air pressure can be only temporary unless the cause of the Eustachian obstruction is removed. It is on this account that the too active use of the bougie is to be condemned, and we have been wont to meet with transient benefit from our practice of bi-weekly or tri-weekly inflation.

II. The discovery of the cause of the Eustachian obstruction, when such exists, is often very difficult. As we have just stated, it is often a question whether in many cases this is to be found in the nose and throat, and it is probable even where it is, that the condition of the nose and throat and of the ear is dependent upon a common cause. It is to be freely admitted that in many instances this cause may not be discovered, while in others, it is quite evident. In not a few cases, auto-infection from the gastro-intestinal canal seems to be the particular excitant. Certainly in many cases of throat trouble this condition is present and is easily discovered if looked for.

For many years rheumatism and gout and scrofula have been regarded as causative factors, while syphilis must never be over-

looked. Inheritance certainly is a weighty agent—Lucæ says—in at least 37 per cent of all cases. It is, however, very questionable if there does not exist in many instances a still more far reaching dyscrasia. The relation of auto-intoxication from the blood, commonly spoken of as uric acid diathesis, to acute and chronic affections of the nose and throat has not been thoroughly defined. We all recognize, however, that it plays an important role, and that the popular impression that colds are the result of exposure, merely, is not correct. In the same way, these poisonous elements of the blood can exert their baneful influence directly or through the medium of the nose and throat upon the ear. Much more investigation and study in this direction is demanded; for above all it should be our aim by every means in our power to discover and check, if possible, the primal factor at work in the middle ear disease; but,

III. Most difficult of all, and yet of paramount importance in the question of prognosis, is the problem, clearly a hopeless one where extensive bone deposit has taken place, of promoting absorption of the pathologic deposit within the tympanic cavity, and the restoring of normal function thereby to the sound conducting apparatus. This is the task for the student of otology. It is a field which has tempted many a bold Roland in the past, who alas, has never reached the "Dark Tower."

Vapors and fluids, both through the Eustachian tube and through the drum-membrane, have been tried and long since given up. Pneumo-massage at one time seemed to offer large promise, but, of late years, has been abandoned by most surgeons except in well selected cases. Sexton, Dench and Jack have urged the value of operative interference, but the result of their work in this direction has not been encouraging. Mechanical and vibratory massage to the Eustachian tube with a view to stimulating the lymphatics and to promoting absorption offers a considerable degree of encouragement and hope. Much has been claimed for climate in an empiric way, but as far as the writer knows, the ideal climate has not been described.

It has been the practice among the more conservative members of the profession to attach an entirely unfavorable prognosis to cases of oto-sclerosis. That this is not entirely justified is shown by the report of Toynbee, in his book published more than fifty years ago, of a number of cases which undoubtedly belong to the early stage of this class, where by the internal use of mercury and

counter irritants to the mastoid, he was able to restore to a reasonable degree the hearing of persons of advanced years. It is in this connection of interest to recall that this father of modern otology felt that the tendency of the German school in the use of the catheter, which was making itself evident at that time was not rational, and that the result, while temporarily satisfactory, could not be maintained unless the producing causes were removed.

More recently the value of massage to the chain of ossicles by means of pressure applied by a specially constructed probe, has been advocated by Professor Lucae and in his latest book he still urges its use in not too far advanced cases of oto-sclerosis. In a case at present under the writer's care, it has markedly improved the hearing. The chief objection is the pain which it is liable to cause. This, however, can be to a large degree overcome by beginning with the mildest force possible of the spring set, say at 100, by the use of a head-rest, by saturating the cotton wrapped over the metal cup with ether and above all by the exercising of the greatest delicacy of touch in its application.

Internal Treatment.—During the last few years considerable interest especially in Europe has been taken in internal medication to meet the last of the three objects for treatment; i. e., absorption of the pathologic deposits. Sugar of Budapest and Siebenmann have been especially active in this direction. In 1904 Sugar recommended thiosinamin and later a combination with salicylic acid, much less irritating, called fibrolysin. This he has used hypodermatically with good results in from 0.3 to 2.3 grm. doses repeated every day. This preparation, prepared by Merck, comes in sealed ampullae, and, introduced under strict aseptic precautions, causes little or no reaction. He regards its use indicated in the hypertrophic form and in beginning cases of otosclerosis when a primary labyrinthine lesion can be excluded and arteriosclerosis is not present. In the writer's hands improvement was noted in one case of a small series where it was tried. If oto-sclerosis is diagnosed of an advanced degree, especially if absence of prolonged bone conduction, raising of the lower limit of the tuning fork and positive Rinne point to primary disease of the labyrinth capsule, the treatment will do no good nor is it to be employed where a condition of arteriosclerosis is detected as thiosinamin acts through the blood vessels. In this connection it is of interest to mention that certain observers claim that arteriosclerosis is commonly met with in cases

of hereditary deafness. In such cases, iodid of potash in connection with suitable mechanical treatment has been used with benefit Sugar recommends as an improvement, a synthetic product called sajodin, a calcium salt containing 20 per cent of iodin without taste or smell. Of this he gives 1 to 3 grammes a day after meals. A still newer preparation is iodipin, which it is claimed, can be used hypodermically without evil effect. One cc. of a 10 per cent solution is injected near the spine from two to seven times a week and increased 2-5-10-20 cc.

Another suggestive relation etiologically and therapeutically is that of disease of the thyroid gland to progressive middle ear deafness. While not a new theory and much combated, Sugar in the article just referred to, expresses himself in favor of it and accordingly is in the habit of giving thyroidin for selected cases of otosclerosis with good results on account probably of the iodin which it contains. Aside from these special remedies, emphasis is again to be put on the importance not merely of a careful functional examination, but upon a thorough physical examination as well, including in many cases that of the blood and urine. As specialists we are liable to underestimate the value of such an examination. A condition of the kidneys may account largely for the trouble in the ear or the constitutional cause just spoken of may be found in the gastro-intestinal tract or in a dyscrasia of the blood. If a history of rheumatism is elicited, that without fail demands treatment, and even if we are unable to discover any such constitutional cause, we should not underrate the importance of careful attention to the general health of our patient. This is equally true whether the ear condition is one of hypertrophy or of sclerosis.

In any case the excessive use of tobacco or alcohol, obstinate constipation or undue nervous excitement or worry are almost sure to aggravate the condition and destroy our best endeavors. Above all, will any acute inflammation of the ear, especially of a purulent nature, prove disastrous to the hearing. To conclude, while a great deal remains to be accomplished and while advanced cases offer but slight hope for relief, rational treatment to beginning cases is capable of accomplishing much not only or chiefly by restoring the former normal status, but by preventing, through discernment of the true condition present, local as well as general, an advance of the pathologic process.

CANDIDATE'S THESIS.

THE RELATIONSHIP BETWEEN DEGENERATE TONSILS AND MIDDLE-EAR DEAFNESS.

J. N. REIK, M. D.

It is the object of this paper, as indicated in the title, to consider the possible effect of the so-called "degenerate tonsils" in the production of middle-ear deafness, and the manner in which they may act in bringing about pathological changes in the middle-ear cavity; not to discuss the effect of those general hypertrophies of the tonsils and lymphoid ring, so commonly met with in childhood, the relationship of which to middle-ear deafness is so well understood and conceded. The cases reported in this communication occurred in adults, who consulted the otologist for the relief of deafness, and who did not complain of any throat or nasal symptoms until questioned regarding recurrent or constant slight pharyngeal irritation or voice impairment, when on careful examination diseased tonsils were found. The fact that the complete enucleation of such tonsils has, in a number of cases, improved, arrested, or even cured cases of beginning or progressive middle-ear deafness and tinnitus, without other treatment, leads to a consideration of the manner in which these tonsils may possibly act in producing such pathological conditions in the ear.

Middle-ear deafness may have its origin in at least three ways: First, it may result from the extension to the tympanum of inflammatory conditions in the adjacent tissues of the Eustachian tube or pharynx; second, it may be due to alterations in the vascular supply of that cavity; third, it may be due to interference with proper and adequate ventilation of the tympanum through the Eustachian tube.

A case seen last week shows very clearly the working of the first named process, a pathological condition which is understood and conceded by all, though in cases similar to this one the originating disease focus may easily be overlooked.

L. G., a boy of five years, was observed December 17th, 1907, with the parents' statement that during the early hours of the morning the child had developed severe earache on the left side, which subsided after a few hours with the use of hot applications. At the age of two, after an attack of middle ear disease, he had been subjected to an operation for the removal of adenoids and tonsils, the guillotine instrument being used. Examination showed the left tympanic membrane bulging in its posterior inferior quadrant under the pressure of probable serous exudate. There was no fever nor pain at the time of examination. Naso-pharyngeal examination showed a remnant of lymphoid tissue over the vault, extending from the orifice of one Eustachian tube to that of the other. On the right side was a medium sized tonsil, projecting somewhat beyond the pillars, while on the left side there was a submerged tonsil, which contained a few white points.

An operation under general anesthesia was performed, the adenoid tissue being removed by curettement, and both tonsils dissected out. The interesting point in this case is that the small submerged left tonsil—on the same side as the acutely inflamed ear—contained a considerable amount of pus, which was discovered only by accident when the tonsil was cut for examination; in other words, it contained an apparently encysted abscess. Paracentesis of the left tympanum evacuated a quantity of sero-purulent fluid.

The tonsils often show the first signs of infection, to be later followed by middle ear involvement. The organisms in the mouth may gain entrance through these tonsils and produce disease of the middle ear just as they cause joint involvement and cardiac disease. Any of the organisms which are generally lurking in the mouth or in these diseased tonsils (usually one of the forms of cocci) may also be conveyed to the tympanic cavity from these tonsillar breeding places by the blood or lymph channels. It is therefore of great importance in every case of ear involvement to give close attention to these glands lest a diseased condition be overlooked, and complete destruction of these foci, by enucleation of such tonsils, should be a preliminary to any other form of treatment. It is hardly necessary to go into detail regarding such examination, but where the patient does not complain of any throat trouble, the otologist is very likely not to make a searching examination of these glands with a probe, and so, very often, such foci of disease are overlooked or ignored, as they can be discovered only by pull-

ing forward the anterior pillar and probing the hidden crypts and recesses of the tonsils.

It is, however, to the possibility of these tonsillar masses acting mechanically to supply the other two causes, vaso-motor changes and impaired ventilation of the middle ear, that I would particularly call attention. The return of blood from the middle ear is in part through veins running superficially through the mucous membrane of the Eustachian tube into the lateral pharyngeal veins, and any mechanical pressure on the lateral walls of the pharynx may unquestionably result in a blood stasis of these small vessels.

Regarding the blood supply of the middle ear, Gruber[1] says: "The middle ear receives its blood supply from the external and internal carotid arteries. The branches specially distributed to it are:

(a) "Small branches from the arteria pharyngea ascendens supply the tuba Eustachii and its muscles, and some penetrate the mucous membrane of the tympanum to supply it."

(b) "The arteria stylo-mastoidea, a branch of the arteria auricular posterior in its passage through the canalis Fallopii, gives off small branches which enter the tympanic cavity through its posterior wall."

(c) "Small branches of the arteria temporalis pass through the Glasserian fissure into the tympanic cavity." He says further, "The venous blood is drained away by veins which accompany these arteries, and which pour it into the venous plexuses near the temporo-maxillary joint, into the veins of the pharynx, and also into the middle meningeal veins."

Mechanical pressure, quite sufficient to prove obstructive to a venous flow of blood, may be readily produced by even a small tonsillar mass, especially if it be in a diseased state, with accompanying oedema of the deeper tissues. It is much more likely to be produced by the degenerate, submerged tonsil than by the simple hypertrophic tonsil or adenoid. It is often evident on careful examination that even though the tonsil does not project beyond the pillars into the throat, and though practically invisible on casual inspection of the pharyngeal cavity, yet on drawing aside the tonsillar pillars or lifting the tonsil out of its bed with tenaculum forceps, a large tonsillar mass lies deeply buried under the pillars, in just the position to exert marked pressure on the lateral pharyngeal walls. The large hypertrophic tonsils so common in child-

(1) Disease of the Ear, Gruber, 2nd Amer. Ed., 1893, p. 87.

hood are not so apt to act in this way, because the bulk of the hypertrophied tonsillar mass in such cases projects into the throat and there is, in consequence, no backward pressure upon the vessels.

When venous stasis in the deep pharyngeal vessels is produced, there follows a hyperaemic condition of the mucous membrane of the tympanum, and if it be continued, a serous exudate into this cavity, with the attendant possibilities of such a condition produced in any other way.

Furthermore, a condition of venous stasis naturally interferes with the reception by the tissues of the normal amount of arterial blood, and consequently there is a trophic disturbance of the tissues that may likewise result in impaired function. Franklin H. Hooper,[2] in his excellent article, written many years ago, on the evil consequences of adenoid growth pointed out the possibility of vaso-motor and trophic disturbances of the tympanic cavity, resulting reflexly from any inflammatory irritation in the naso-pharyngeal region, and Politzer,[3] who believes he has proven a close anastomotic connection between the vessels of the middle ear and those of the internal ear, says: "This relation of the tympanic mucous membrane to the osseous wall is of special importance inasmuch as inflammation of this membrane can produce transitory or permanent hyperaemia, and disturbances of nutrition in the bone and in the labyrinth."

The third mode of action and the one to which I would most especially direct attention is that whereby the tonsillar mass may interfere with the function of the Eustachian tube. Under normal conditions this tube is patent, but not open, its superior-posterior lip lying in contact with its anterior-inferior. It is regularly opened for the admission of air to the tympanum in certain involuntary muscular actions, such as the process of deglutition, and the making of certain sounds in phonation. This act of opening the orifice of the tube is accomplished by the delicate mechanism of the tubal and palato-tubal muscles; the tensor palati, levator palati and dilator tubæ. During deglutition the soft palate is raised and put upon the stretch by the levator palati and the tensor palati muscles. The lowest muscular structure of the soft palate is the palato-glossus, arising from the side of the posterior part of the tongue and running upward to the under surface of

(2) Boston M. & S. Journal, 1886, pp. 193-7.
(3) Diseases of the Ear, Politzer, 1903, p. 37.

the soft palate, where it spreads out and becomes continuous with that of the opposite side; this forms the anterior tonsillar pillar and aids in promoting the tension of the soft palate in the act of swallowing. The palato-pharyngeus forms two muscular strata in the soft palate, enclosing between them the levator palati muscles, these two strata coming together outside the soft palate to be joined by two delicate muscular slips which spring from the lower border of the cartilage of the Eustachian tube, and are sometimes described as the salpingo-pharyngeus muscles. Spaltcholz refers to these as the salpingo pharyngeus, and says that they are attached to the lamina medialis cartilaginis tubae auditivae. The palato-pharyngeus thus formed, and intimately connected with the tube, arches downward and backward as the posterior pillar of the tonsil, to be inserted into the thyroid cartilage, some of its fibres extending backward to be inserted into the pharyngeal aponeurosis.

The levator palati, which, as has just been said, is enclosed between the layers of the palato pharyngeus, is a round, fleshy muscle arising from the lower, inner border of the cartilage of the Eustachian tube.

The tensor palati, which lies in front of the levator palati, is flat and band like, arising from the scaphoid fossa, from the outer aspect of the Eustachian tube, and ending in a tendon which turns under the hamular process; in the soft palate this tendon expands below the deep part of the palato-pharyngeus (posterior pillar) to be inserted into the transverse ridge of the palate bone.

A muscular slip, which descends from the outer margin of the cartillage, in relation to the outer, unprotected side of the Eustachian tube, and which joins the tensor palati, has been termed by Rudinger the dilator tubae. The palatal aponeurosis extends backward from the posterior margin of the palate, and a portion of the deep fibers of the palato-pharyngeus, some of the fibers of the levator palati, and the tendon of the tensor palati have attachment to it.

It may be seen from this consideration of the mechanism of the Eustachian tube muscles that their action is intimately connected with that of all the palatal muscles and with the so-called tonsillar pillars, and anything that interferes with the normal act of deglutition may affect the palato-tubal function.

Is it not conceivable, then, that interference with the action of these muscles may be occasioned by the presence of a tonsillar mass

adherent to or causing a low grade of inflammation of the tonsillar pillars, and may not the improvement in the auditory conditions following the complete enucleation of these masses be due sometimes, or in some measure, to a freeing of these muscles?

Francis H. Hooper, in his article referred to above, says, "Finally, these adenoid growths may cause paresis of the palato-tubal muscles, and thus be a factor in the production of so-called 'paretic deafness.'" It would seem to me that this statement might be applied as well to these tonsillar masses.

Blake and Reik in their recent book[4] say "that the ventilation of the ear through the Eustachian tube is assured by the action of the lavator and tensor palati muscles which are attached to the soft palate at one end and the anterior walls of the tube at the other, and that contraction of the palatal ends of these two muscles causes a corresponding movement at the tubal ends, the result being a withdrawal of the interior from the posterior wall of the tube, and at the same time a depression of the inferior wall or floor of that passage, giving it a greater inclination downward towards the pharynx. The same movement which opens the tube tends to compress the air in the naso-pharynx, thus favoring still further the ventilation of the middle ear." They also say "that with the presence of adenoid growths in the naso-pharynx this admirably working mechanism is interfered with, even when the growth is not large enough to occlude the orifice of the tube, as it interferes with the palatal movements and with the proper balance of air pressure in the naso-pharynx. Even a small growth may thus interfere with the palatal movements, as is shown by the effect which it has upon the pronounciation of the nasal consonants, that is to say, upon the palatal movements." This is certainly equally true of these tonsillar masses.

It would seem to me that in most of the cases to be referred to there was not sufficient inflammation of the tonsils to alone account for the aural symptoms. In fact, in only one, which has been already related, was this explanation applicable.

We know that hearing is diminished whenever the air pressure within the tympanic cavity is allowed to become less than the external atmospheric pressure, and the pressure within can only be kept the same as that of the outside atmospheric pressure by the regular uninterrupted opening of the pharyngeal tube orifice. If

(4) Surgical Pathology and Treatment of Diseases of the Ear, 1906, pages 288-292.

the tube is kept closed the blood vessels of the tympanic wall take up the oxygen from the imprisoned air and give off carbonic acid gas in exchange, but the amount of the latter given out is less than the amount of the oxygen removed, so that the total quantity of gas within the closed cavity is reduced and its pressure consequently becomes less than that of the atmosphere—the membrana tympani is cupped inward, and the increasing tension of the membrane may occasion deafness or dulled hearing, before any exudative condition within the cavity has occurred. If this state of affairs continues an exudative inflammation is soon started up, and if this cause, or such exudative inflammation, can be removed before nature has transformed the exudate into embryonic or adult connective tissue, graver consequences may be prevented.

The operative procedure in the cases to be related was that of separation of the tonsillar pillars and complete enucleation of the tonsils, with dissecting forceps and a sharp bistoury, thus leaving the muscular pillars clean and free to perform their function.

The ordinary tonsillotomy, even in such cases as might admit of that operation (that is, if the tonsils projected beyond the pillars), would not act in this manner, of course, as there would still be left a tonsillar stump, besides which the injury to the delicate muscles of the tonsillar pillars attending such operation would still further hamper their action, instead of improving it. Moreover, it is the tonsils which, instead of projecting into the throat, extend backward behind the pillars, the so-called submerged tonsils, that are encountered in these cases, and nothing but their complete enucleation is indicated, and, of course, nothing short of that could be expected to have any influence on the conditions described. Furthermore, even in the cases of large hypertrophied tonsils, in patients already predisposed to pharyngeal deafness, tonsillectomy by dissection is to be recommended, because tonsillotomy by the crude method of the guillotine may injure the delicate muscle slips running in the tonsillar pillars, and thus still further weaken the already defective action of the palato-tubal mechanism.

Though the aid of intact and perfectly functioning tonsillar pillars may not be absolutely necessary to a proper functioning of the tube, in all cases, yet in certain cases of weak tube action just this additional aid may be needed. Inflation of the middle ear by means of Politzerization and catheterization is of only temporary

benefit in these cases. It should be our aim to restore to the tube its function of properly opening itself.

The following cases are a few of those accurately observed, and serve to illustrate the relationship referred to:

Mr. H. L. of Wisconsin, first examined April 3, 1907, because of subacute otitis media of recent origin, accompanied by pronounced deafness and sense of "stuffiness" in both ears. His condition had followed an acute naso-pharyngitis, but he had been subject to recurrent attacks of slight otitis for the past year or so.

Examination of the ears showed a hyperemic condition of both membrani tympani, with probable serous exudate in the cavity. Gentle catheterization relieved the condition temporarily. He had degenerate submerged tonsils, which had not been giving him any trouble.

Hearing tests showed: Watch R. and L., c.40; bone conduction, R. and L. 15.15; air conduction, 10.30; Weber equalized.

On May 1, one week after a complete tonsillar dissection, and without any aural treatment after the first visit, his hearing had improved to ability to hear ordinary conversation, as against only very loud conversation before. Watch, R. 20.40, L. 8.40; air conduction, R. 20.30, L. 15.30. At that time he left the city, but has been heard from within the past month, and is reported to have perfect hearing, with no complaint of aural or pharyngeal trouble.

Mr. D. L. W., first examined April 9, 1907, complaining of long standing deafness in the left, and recently increasing deafness in the right ear. He believed his deafness in the left ear had been congenital. Aural examination showed the right tympanic membrane slightly retracted and hyperaemic. During the past few months his deafness on the right side had shown a marked tendency to increase, and it was becoming difficult for him to hear ordinary conversation or to enjoy public lectures. The changes in the tympanic membrane were slight, but he had a small, ragged, submerged, bad-looking left tonsil, and a very large right tonsil, which was adherent to the tonsillar pillars.

Hearing test showed: Watch, R. 20.40, L. 0.40. Low tone limit, R. (c. 64 v. s.); L. (c. 256 v. s.); bone conduction, R. and L. 10.10; air conduction, R. 15.20, L. 7.20; Weber lateralized to left.

A double tonsil enucleation was advised and operation performed April 14. On April 22 hearing test showed: Watch, R. 30.40, L. c.40; air conduction, R. 18.20, L. 10.20.

In the latter part of May a further test showed that the left ear maintained the same condition, while the right had improved to normal hearing.

Miss S. E. T., Ellicott City, seen May 24, 1907, complaining of increasing deafness in the right ear and a chronic deafness in the left ear which had existed for four years. Both membranae tympani presented the appearance of chronic exudative otitis media, slight retraction and thickening, and hearing tests showed marked interference with the transmitting apparatus.

Air conduction (c. 512 v. s.), R. 13.20, L. 15.20; watch, R. 15.40, L. 20.40; Weber equalized. Low tone limit (c. 64 v. s.).

She had a slight hypertrophy of the right inferior turbinate and submerged, degenerate tonsils, with white, cheesy material in the crypts. Complete tonsil enucleation was performed on June 1.

Unfortunately no record was made of her hearing tests immediately after. She went away for the summer, had no treatment whatever, but returned on October 9, to report that she had been perfectly well in every respect, and hearing tests at that time showed: Watch, R. and L. 30.40; air conduction, 20.20.

I have seen her within the month and she declares that her hearing is perfectly normal.

Miss B. H., Washington, aged 33. First seen October 1, 1907. She came for relief from deafness, tinnitus and occasional attacks of throat inflammation, which she said had existed in some degree for three years.

The right tympanic membrane was thickened, slightly retracted, and the left one markedly so. She had tried various drugs and forms of aural treatment, including a course in Christian Science. Ordinary conversation was heard with great difficulty, and only in a quiet room.

Hearing tests showed: Watch, R. and L. 0.40; low tone limit (c. 128 v. s.); bone conduction, 10.10; air conduction, R. 10.20, L. 7.20.

Pharyngeal examination showed a degenerate submerged left tonsil of fair size, and a smaller but more degenerate one on the

right side, the pillars and tonsil on this side having the appearance of being all matted together. Nasal condition was normal; large but not obstructive inferior turbinates.

A double tonsil enucleation was done on October 3. Her hearing improved in both ears, and on October 15 hearing test showed: Watch, R. 4.40, L. 0.40; air conduction, R. 19.20, L. 14.20; whispered voice at six feet; conversational tone at 18 feet. The tinnitus, she said, was scarcely noticeable, though still existing to a slight degree.

Mr. B. L., North Carolina, seen December 6, 1906, because of a slight loss of hearing in the right ear. He complained also that there was a sense of "stuffiness" in that ear, with occasional attacks of pain. The condition had existed for some months, with various periods of improvement and recurrence, and was becoming quite annoying.

Aural examination showed a subacute otitis media, the tympanic membrane being congested about the periphery particularly and slightly retracted. Hearing test: R. E.: Upper tone limit normal; lower tone limit (c. 64 v. s.); air conduction, 15.20; bone conduction, 10.10; Weber referred to right.

Examination revealed small submerged, degenerate tonsils on both sides. With the Eustachian catheter air could be introduced into the tympanum, but with some difficulty and with only temporary improvement. His engagement as a student prevented operative procedure at once, and he was treated with sprays and applications and catheterizations for some weeks, when he submitted to operation.

Almost immediately the aural affection, which had persisted all the time, disappeared, and this result has been permanent, for a report from him within the month says that he is perfectly comfortable, and that there has been no recurrence of the ear trouble; that his hearing is normal.

Mr. J. D., Ohio, seen January 22, 1907, complaining of irritable throat and dullness of hearing. He said that his throat was more or less constantly sore, that his voice failed after a very short period of use, and that his friends had begun to complain of his impaired hearing. Hearing tests: Watch, R. E. C.40, and L. E. 140; whisper, R. E. 8 feet, L. E. 10 feet; acoumeter, R. E. 10 inches, L. E. 15 inches; bone conduction normal; air conduction,

R. E. 10.20, L. E. 12.20; upper tone limit normal; lower tone limit (c. 128 v. s.); Weber not lateralized.

On examination of the throat and nose there was found small diseased, submerged tonsils, an elongated uvula, and a hypertrophied inferior turbinate on the right side. The tonsils were dissected out and the uvula clipped. Four days later, when I next saw the patient, there was marked improvement in his ability to hear the voice, and he stated that he could hear much better. There had not at any time been any notable alterations in the tympanic membrane, so that no changes could be observed there, but his hearing now rapidly improved until his air conduction for the tuning fork (c. 512 v. s.) became 20.20, and he could hear the whispered voice at 20 feet and the watch at 15.40. This was without any treatment whatever directly to the ear.

In October he reported through a friend who was coming to Baltimore that he is perfectly well, can use his voice all day without tiring, and hears quite as well as anyone.

Miss G. E., Mt. Washington, first seen January 28, 1907, complaining of recurring attacks of earache, involving the left ear particularly. The left tympanic membrane was hyperemic at the time, the membrana flaccida being most congested, while the membrana tensa was retracted. There had been considerable pain during the night before and periodic attacks of soreness for a week. Both tonsils were large, inflamed and adherent to the faucial pillars. Her mother desired postponement of operative treatment for a while in order to permit completion of some school work, and consequently local antiseptic and astringent applications were employed until the month of March.

At that time hearing tests showed: R. E. and L. E. watch, 20.40; bone conduction, R. E. and L. E. 10.10; air conduction, R. E. 10.20 and L. E. 8.20; Weber not lateralized. On March 4 both tonsils were enucleated and a small adenoid removed. No treatment to the ears was instituted until March 20, when the hearing had markedly improved, air conduction for the fork having increased to 15.20 for the right and 12.20 for the left, while the watch could be heard at 30.40 in each. Politzerization, the use of chloroform vapor and massage caused a further improvement, and on April 10 air conduction was R. E. 20.20 and L. E. 18.20, a condition which she retains at the present time without any further treatment. The gain of 50 per cent in auditory function with-

in two weeks after tonsillectomy and without any aural treatment is striking.

From a study of these cases, seen in private practice, and others of a similar character treated in the dispensary, the importance of carefully looking for these diseased glands, and getting rid of them completely when found, in all cases of beginning or progressive middle ear deafness, has been impressed upon me; not that the enucleation of such degenerate tonsils will always arrest the progress of or cure the condition, but that it will eliminate at least one of the possibly important factors in the production of such condition.

PAPER:

MEMBRANOUS PHARYNGITIS COMPLICATING INFLUENZA.

JOHN F. WOODWARD, M. D.

Correct therapeutics should follow correct diagnosis. A recognition of the true pathology of a case is necessary to a correct diagnosis. The following cases will illustrate:

Case I.—Miss C. E., of Norfolk, Va., sent for me some time in January, 1905, saying that she was too ill to go to my office. I found her in bed with a severe attack of grip—temperature 102° F.—Her family physician had seen her for the previous week and reported an unusual severe attack of influenza—especially the throat symptoms. Great masses of whitish membrane hung from the naso-pharynx, which I removed with cotton swabs, upon the removal of which there appeared beneath fully organized, tough membrane extending from about midway the posterior pillars up to the fossae of Rosenmuller tightly adherent to the crater-like tops of a swollen chain of glands. Upon tearing this membrane away with forceps there was decided bleeding. The rest of the pharyngeal space was full of swollen glands, but these were very sparsely covered with membrane, though very much congested. The glands

of her neck, especially those directly under the ears, were very much swollen and very tender to the touch. The tonsils took very little part in the surrounding inflammation. A tendency to chilly sensations and very great pain in the upper throat was coincident with the swelling in the glands of the neck. This indicates that there must have been very decided absorption of toxic material at this time. The impression upon the nervous system when I first saw her was very profound, more than in an ordinary case of influenza. I immediately had the membrane examined under the microscope, and the report was negative, except that large quantities of streptococci were found. After cleaning the throat of the membrane I examined the rhino-pharynx closely and found that the exudate did not extend into the nose at all. Her family and personal history were all that could be desired. It seemed impossible for her to hawk up the membrane—it had to be pulled away from the tops of the glands with forceps. My treatment was entirely local as her physician had very wisely put her on an excellent treatment.

Case II—Mrs. H., of the same city and same street as above, consulted me in February, 1906, with a similar condition, with the exception that the age of the patient was very much against her, being about 75 years of age. The nervous prostration that accompanied her attack was very alarming. In this case there was a very decided irritation of the nasal mucous membrane, but no membranous deposits in any part of it, only a free discharge of mucous.

Case III.—Occurred in the same month and same year, and showed no special features over the others.

Cases IV, V and VI have all been seen this year since October 1st. They exhibit no special features, except that they have all been in women. Two of them on the same street and within two blocks of one another. One in a lady of about 70, where the poisoning was very profound and the convalescence prolonged to about two months.

I have been treating cases of acute pharyngitis for the last sixteen years, but not till 1905-6-7 have I made notes of special features in these cases accompanying influenza. If I have ever treated such cases before it was faulty diagnosis to record them as simply acute pharyngitis. The profound impression made upon the nerv

ous system, the evidence of rapid absorption of some toxic material seen in the swollen glands, rise of temperature and the great pain in the upper throat, as well as the nature of the membrane would lead us to look upon it, if not as a separate and distinct condition, certainly as an unusual variety of acute catarrhal pharyngitis.

Having the anatomy of the rhino-pharynx clearly in mind you readily see the necessity of recognizing the exact nature of any acute formation of membrane in that locality. Note the chain or ring of lymphatics radiating therefrom, and you are not surprised that disinfection of the rhino-pharynx is now being advocated as a precautionary measure as well as a treatment in cerebro-spinal meningitis. The Pfeiffer bacillus has been found in the cerebro-spinal fluid. In fact, the long list of ailments that seem, in some way, to have had a primal origin somewhere in the tonsillar triangle is attracting as much attention to-day as the opsonic index or the Great White Plague.

An article by Schadle on Membranous Laryngitis Complicating Influenza, recently published, called to my mind these cases of membranous pharyngitis. Call it what you wish, but when you see a case you will not be in a hurry to tell your patient that she has simply a case of acute pharyngitis. I saw none of these cases in their first stages, it was always after a few days of influenza, therefore I cannot say what was the first pharyngeal symptom. Not one of them was subject to gout or rheumatism, and their environments were all that could be desired, in fact, all were in ladies of refinement who lived in comfort. I have not seen a single case in a male patient. In each case the membrane was put under the microscope, and every case proved negative, only streptococci were found.

I was called to the home in each case with about the same history. There had been a day or so of sore throat accompanying an attack of influenza. The onset of the membrane formation seems to have been the signal for pain in the naso-pharynx, fever of from 102° to 103°, and swelling and pain in the glands of the neck, especially those just under the ears. The pain in the pharynx seems to have been most severe whenever an effort was made to clear out the membrane by hawking.

There was found about the same conditions in each case, tonsils and posterior pillars slightly congested, but taking no active part

in the general condition. Long strings of whitish membrane hung from the vault, which, when removed, revealed large rolls of white membrane beneath closely attached to the cup-shaped tops of a chain of swollen glands extending on either side of the pharynx from the middle of the posterior pillar to the fossa of Rosenmuller. The rest of the pharynx was granular and red, but seemed to take little part in the formation of the membrane. When pulled away with forceps the crater-like tops of the glands bled rather freely. Two cases had earache, one had rather free discharge of mucous from the nose. In neither one of the cases did the membrane originate in or extend into the nasal cavities. Two of the cases in ladies about seventy years of age suffered great physical and mental depression. The younger patients had greater amount of pain, and in all of the cases the grip symptoms, pain in joints, headaches, general malaise, etc., disappeared with the membrane.

These cases have been of unusual interest to me, if for no other reason than that of diagnosis. Whilst the microscope did not find the Klebs-Loeffler bacilli, streptococci were found, showing that the condition was not entirely free from danger. The great amount of pain, the high fever and great physical depression that existed from the time the membrane appeared until it subsided, along with the symptoms of rapid absorption of toxines seen in the cervical glands, were the special features that seemed to differentiate it from simple acute catarrhal pharyngitis.

PAPER:

CONTOUR AND CONTENTS OF THE EPITYMPANUM IN RELATION TO SUPPURATIVE DISEASE.

CLARENCE JOHN BLAKE, M. D.

The epitympanum while varying greatly in size and shape in different individuals, still possesses certain uniform characteristics which are a guide in the determination of the nature of remedial measures, applicable to diseased conditions of its contents.

For purposes of classification the epitympanum proper may be regarded as a very nearly circular cavity slightly compressed from within outward, and from below upward, its major portion lying posteriorly to the median vertical line of the tympanic cavity, and its walls, in contradistinction to the floor of the tympanum, and the inner wall of the aditus ad antrum mastoideum, being of a smooth and even contour.

The downward and inward slope of the outer epitympanic wall serves to make the bony portion of the epitympanic floor, the remaining space between this and the opposite tympanic wall being partly filled in by the upper surface of the short process of the malleus and the attachments between this bone, the incus, and the outer epitympanic wall, and bridged over by the folds of mucous membrane, with occasionally interposed laminae of fibrous tissue, which are found, to a greater or less degree, in more than 85 per cent. of normal human temporal bones.[1]

It is this main cavity of the epitympanum which is the most constant in its position, size and outline, the greatest variations being those which concern the extensions from this cavity backward and upward in the form of varying recesses in, or encroachments upon, the surrounding diploetic space.

(1) Reduplications of mucous membrane in the upper portion of the tympanic cavity. Boston Med. & Surg. Jour., May 30th, 1889, 531. C. J. Blake and W. S. Bryant.

In some instances these encroachments leave a multiple layer of diploetic cells between the tympanum and the superficial cortex of the temporal bone, either inward toward the pyramid, upward toward the tegmen tympani, or backward toward the mastoid, while in still other cases the recesses, apparently joining in the formation of one large pneumatic space, have made the outer cortical layer the only boundary between themselves and the cranial cavity.

With such diversity of contour and such variety in approximation of the superior epitympanic space to the cranial cavity, the question of the speedy outcome of a suppurative process in the epitympanum, in so far as its lethal extension is concerned, would seem to be a matter of considerable dubiety.

It is not, however, until the contents of the epitympanum is made the subject of a careful study, and the possible protective limitation afforded to the progress of a suppurative process is appreciated, that an assurance is apparent, the knowledge of which can be utilized in the prolonged treatment of epitympanic suppuration.

The central, circular, portion of the epitympanum enclosing the larger portions of the major ossicles, bounded posteriorly by the extremity of the recessus incudis and inferiorly by ligamentous attachments and supplementary ligamentous bands, with the addition of mucous folds, becomes almost a cavity by itself, its limited means of communication, with the adjacent parts of the tympanic cavity, being speedily closed in the event of engorgement of the lining membrane.

This engorgement of the mucous, and submucous, tissue, forming the lining of the tympanic cavity throughout, varies greatly in different parts of the cavity and is demonstrable in cases of large perforation, or destruction, of the drum-head, and in cases of destruction of the outer epitympanic wall, the known vascularity of the lining membrane of the upper and posterior portion of the epitympanum favoring a rapidity and persistence of engorgement sufficient not only to compress the retained air in the lower portion of the tympanum and press the drum-head outward, but also to afford a prolonged closure of the aditus and thus still more effectively comprise the epitympanum of pathologic causation, within the boundary limit, of what may, anatomically, be called the

epitympanum proper, the constant space factor, in contradistinction to the extremely variable, and therefore inconstant, space factor beyond it.

Within such an encapsulation a congestive or suppurative process may run its course, its apical outlet being outward and downward through the pars flaccida or through the posterior portion of the outer boundary of the epitympanic space, into the tissues of the upper portion of the membrana vibrans, the remaining portions of the tympanic cavity remaining untouched. Examples of this are to be found in the cases of free serous exudation through the upper portion of the drum-head, accompanying acute congestion, and in the cases in which a carious process of the head of the malleus has run its course to destruction without lateral, or inferior, extension of the suppuration, or other drainage than that afforded by the opening in the pars flaccida. In the initial congestive stage the swelling of the mucous folds, and of the constant mucosa of the aditus, tends to protect not only the underlying diploe, there so extensively present, but to block the major passage backward into the mastoid through the pneumatic chain, of which the epitympanum is the initial member, while in the more advanced suppuration not only the engorgement, but also the proliferative swelling of the soft tissues, serve the uses of limitative protection, and, even where a carious process has been initiated in the major ossicles, or on the outer epitympanic wall, the tendency is to wall the area off from the surrounding healthy structures by engorgement, proliferation, or the production of reparative granulomata.

Examination of the supplementary ligamentous attachments of the ossicles, as well as of the epitympanic mucous folds, shows a tendency to favor drainage of the epitympanum, outward either over the incline of the short process of the malleus or through the posterior portion of the membrana vibrans, in its upper segment, the anterior rounded border of the epitympanic space being above the incline of the tympano-pharyngeal tube and its posterior, antral, portion terminating on a line with the porterior border of the recessus incudis.

The horizontal folds are usually two, sometimes three, in number, one stretches from the anterior border of the tensor tympani tendon forwards and outwards, attaching itself to the

anterior ligament of the malleus, and to the anterior wall of the tympanum just above the entrance to the tympano-pharyngeal tube. Another fold covers over the space between the external ligament of the malleus, the posterior ligament of the incus, the incus, and the external tympanic wall. The least common horizontal fold stretches between the long and the short processes of the incus, the posterior crus of the stapes, and the inner wall of the tympanum.

The vertical folds may be considered as two in number, both suspended from the tegmen tympani; one stretching between the anterior and the suspensory ligament and the other between the suspensory and the external ligament of the malleus. The latter fold is often V-shaped, one arm joining the external ligament, the other arm going obliquely outwards and downwards, joining a horizontal fold near the base of the short process of the incus.

Attached to the posterior side of the suspensory ligament is sometimes found a falciform fold, stretching from the articulation of the malleus and incus, backwards and upwards, onto the tegmen tympani.

The space above the pars flaccida is especially rich in mucous folds. These fill the spaces between the ligaments of the malleus in such a way that there is usually enclosed an irregular pyramidal space with its apex at the suspensory ligament of the malleus. This space is bounded below by the malleus, the pars flaccida, and the external epitympanic wall; above by the soft tissues and mucous reduplications below the tegmen tympani; externally by the upper portion of the outer tympanic wall, and internally by the head, and the anterior ligament of the malleus, and by the vertical fold of membrane stretching between the anterior and suspensory ligaments of the malleus; while it is bounded posteriorly by the external ligament of the malleus and the vertical mucous fold between this and the suspensory ligament.

This space is lined with mucous membrane and often crossed by mucous folds; at its lowest point the pars flaccida meets the short process of the malleus on its upper surface, so that fluid within this space gravitates onto the lower part of the pars flaccida.

The results of the examinations of temporal bones show that in 85 per cent. the space immediately above the pars flaccida is

almost wholly enclosed by mucous folds, while in 77 per cent. the epitympanum is partially cut off from the tympanic cavity below it by mucous folds stretching across at least one-half the space.

These enumerated folds, moreover, do not include the reduplications partially bridging recesses in the superior epitympanic space and encroaching upon the lumen of the antrum, folds which, with the more constant mucous reduplications proper, may be safely estimated as more than doubling the vascular surface of the tympanic cavity as a whole.

Investigation, either by section or by transillumination, of the bony wall forming the outer and, partly, the inferior wall of the epitympanum, and coincidently the inner end of the superior wall of the bony external auditory canal shows the reason for the comparative frequency of the spontaneous destruction of this portion of the epitympanic wall in the course of a prolonged suppuration in the epitympanum, with osseous necrosis, where the encapsulating soft structures, engorged and often adherent, have limited extension of the suppuration upward and facilitated the bony destruction downward which most favors the enlargement of the drainage exit from that important direction the posterior extension of the epitympanic space.

In cases of caries of the head and neck of the malleus the destruction of the pars flaccida, and in carious implication of the incus, perforation of the posterior segment of the membrana vibrans, may afford, in themselves, adequate openings both for drainage and for surgical approach, but both drainage and repair may need to be further facilitated by the division of mucous folds and of pathologic adhesions within the area which constitutes the epitympanum proper, as distinguished from the surrounding epitympanic space, and a necessary preliminary to a successful treatment of a limited necrosis within the epitympanum may be not only the removal, by morcellative curettage, or by dry scrubbing, of redundant obstructive granulomata but also the division of folds and bands between the major ossicula and the outer epitympanic wall. This may be easily done by means of the usual angular middle-ear knives, by the curved spatula knife or by the broad bladed mirror knife, cutting being preferable to tearing with a blunt instrument, a ring curette for example, on account of the lesser liability to denudation of bony surfaces still protected and nourished by their normal soft tissues.

Figure 1.—Vertical section of canal and tympanum, showing definition of epitympanic space.

Figure 2.—Antero-posterior section of epitympanum, showing smooth-walled depressions.

PLATE I.

Plates to accompany paper of C. J. Blake.
"Contour and Contents of the Epitympanum."
The Laryngoscope, August, 1908.

Figure 1.—Vertical section of canal and middle-ear cavity, exhibiting proportion and relationship of epitympanum to tympanum, with soft tissues in place.

Figure 2.—Vertical section of middle-ear cavity along manubrial line, showing demarcation of epitympanum by horizontal folds.

PLATE II.

Plates to accompany paper of C. J. Blake
"Contour and Contents of the Epitympanum."
The Laryngoscope, August, 1908.

Figure 1.—Vertical section of an elevated epitympanum bounded externally by large radiate diploetic cells.

Figure 2.—Posterior space of elevated epitympanum with large radiate diploetic cells.

PLATE III

Plates to accompany paper of C. J. Blake
"Contour and Contents of the Epitympanum"
The Laryngoscope, August, 1908.

Figure 1.—Epitympanic space with long transverse diameter.

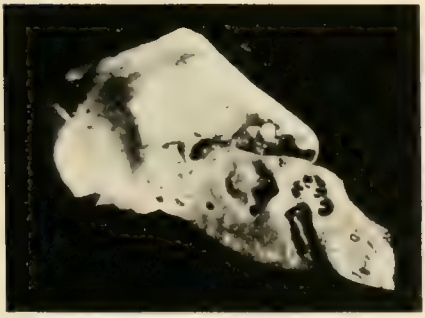

Figure 2.—Horizontal section, displaying recessus incudis and inward curve of antral wall.

PLATE IV.

Plates to accompany paper of C. J. Blake.
"Contour and Contents of the Epitympanum."
The Laryngoscope, August, 1908

Figure 1.—Membrana tympani, malleus, incus and outer epitympanic wall, photographed by transillumination, direct.

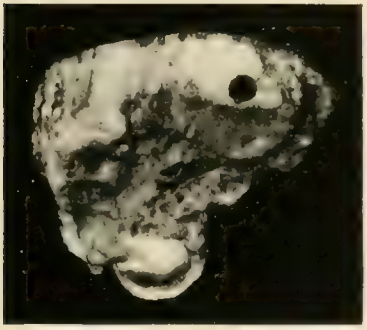

Figure 2.—Membrana tympani, malleus, incus and outer epitympanic wall, photographed by transillumination from below upward.

PLATE V.

Figure 1.—Vertical section exhibiting smooth outer epitympanic wall with diploetic boundary, superiorly and posteriorly, and section of diploetic mastoid.

Figure 2.—Vertical section of large posterior epitympanic space.

PLATE VI.

Plates to accompany paper of C. J. Blake:
"Contour and Contents of the Epitympanum."
The Laryngoscope, August, 1908.

The not infrequent clinical presentation of the results of an old suppurative middle ear process, with the membrana vibrans intact, or showing a conglutinate posterior perforation, with an opening above the short process of the malleus, including a portion of the bony outer epitympanic wall, bounded by involuted cicatrices, dry, clear of congestion and with a fair amount of hearing, is an indication, in what nature has spontaneously effected, of an advisable course of surgical procedure, sufficiently prolonged and patiently pursued.

In the light of structural study and clinical experience the differentiation between the cases of epitympanic suppuration which may be safely treated through the natural channel and those demanding the creation of a new avenue of approach, should be most carefully made and determination should be based upon the merits of the individual case and not upon any merely chronometric estimate.

PAPER:

THE CLINICAL TREATMENT OF INTRACRANIAL DISEASES OF OTITIC ORIGIN.

J. A. STUCKY, M. D.

Some of the members doing the best of otological work who are living in small cities remote from great medical centers, are the ones who need counsel, discussion, and decision from Section meetings like this. The discussions in these Sections for these reasons are often more valuable than the subject matter presented by the essayist, hence the desire for few and short papers, and much and pointed discussion.

I have traveled across four States not with the expectation of presenting something new and valuable to you of the Eastern Medical Meccas, but for the purpose of presenting some lessons learned from individual clinical experience.

We assume that all agree that every case of intracranial complication of otitic origin demands both surgical and medical treatment of most heroic and accurate nature, and this must be given promptly. As to the nature and technique of the operation, we are agreed, but as to the "when to operate" there is much indecision.

The number of cases which I have observed and whose histories have been recorded, is twenty-four, and each case was characterized by marked meningeal and cerebral symptoms, which operation revealed were of otitic origin or complication. They were not of that class, so often found in the mastoid operation, which had not previously given any symptoms of intracranial lesion. Of this number seventeen of the patients were operated upon, ten of whom recovered. In seven others, all the symptoms were improved by the operation, for from one to five days; three cases were meningism as described by Chevalier Jackson, which were of otitic origin.

In the seventeen cases operated upon, one or several of the following conditions were found:

Two cases of abscess of the spheno-temporal lobe.

Five cases of extradural abscess in the middle fossa.

Four cases of extradural abscess in the inferior fossa.

Three cases of serous meningitis in which dark colored fluid was found under considerable pressure between the bone and dura in the middle fossa.

Eleven cases in which the tegmen tympani and antri were destroyed and the dura thickened (pachymeningitis).

Six cases in which the sinus was exposed; two were opened in removing the granulations covering them.

Two cases in which the facial ridge was eroded, exposing the nerve, temporary palsy resulting.

Five cases in which there were fistulous openings in the tegmen tympani.

Six cases in which there was optic neuritis (in both the spheno-temporal abscess cases, and in four cases of extradural abscess in the inferior fossa).

Four cases were due to acute otitis media.

Thirteen cases were due to a chronic otitis media.

In none of these cases was there a lateral sinus thrombosis or cerebellar abscess. Seven of the patients died without operation, and seven died who were operated upon. Of the latter, I was able to obtain autopsies in six, two of which were tubercular meningitis, with the center of infection over the area of the tegmen tympani of a chronic suppurative otorrhea; three were diffuse purulent meningitis, one was diffused meningitis with suppuration of the Gasserian ganglion complicating double mastoiditis. Since operations on intracranial lesion has proven to be life saving, the interest of the otologist and general practitioner has become more active in these patients.

Symptoms of the several intracranial complications of otitic origin in the initial stage are similar, and it is impossible wthout submitting the patient to the danger of delay of deeper and more extensive infection by waiting for focalizing or pathognomonic evidence. For this reason emphasis should be placed on the danger, especially of chronic suppuration of the middle ear, extending to the cranial cavity and sinuses of the dura, and at the first evidence of meningeal irritation, as manifested by persistent headaches, vertigo, vomiting, somnolence or irritability, the radical operation should be

performed, the dura exposed, examined, irrigated and drained through the tegmen tympani and antri.

It matters not whether there exist symptoms of mastoid infection if the middle ear shows evidence of infection, or there is a history of chronic suppuration having existed, I believe it is best to advise immediate operation.

In some of my most pronounced cases of intracranial disease, no pus was found at the operation and no evidence of active trouble existed in the tympanic cavity at the time, but the tegmen of the antrum, attic and tympanum were covered with granulations and the bone was soft and soggy, and gave the sensation more of cutting through wet chalk than through bone.

I am convinced as pointed out by Körner, that in the early stages of infection of the basal meninges, inflammatory edema may produce the same symptoms as observed in the extradural abscess. In three of my patients who recovered extradural abscess of the tegmen associated with compression of the overlying temporal lobes and the cerebral injection was disproportioned to the extradural lesion. While there was in these cases no recognizable abnormality of the intervening arachnoid, by mere contiguity without plastic exudate, the infection had passed through and red softening had begun, which in a few more days would have formed a spheno-temporal abscess. In such cases early operation in which free drainage is given and the infected area thoroughly irrigated, gives promise of favorable results.

The five extreme cases in which the patients recovered were almost moribund. One from home was carried on a stretcher in baggage car sixty-two miles, another thirty-eight miles, the third eleven miles, two others living within a few blocks of the hospital, these cases add increasing emphasis and hope to the plea for early operative interference in the class of cases under consideration, and I quite agree with Randall and others in the contention that in these cases, and also those of tuberculous meningitis the patients ought to be operated on and irrigation with warm physiologic saline solution practiced. In only three cases have I used lumbar puncture, and I am not sure that appreciable benefit resulted. In cases of serous meningitis, I shall continue to resort to it, but am convinced that drainage and irrigation through the middle fossa is the better treatment. I have washed away quantities of purulent exudate in

some of my fatal cases, and though too late to avail more than temporary relief, I am sure the procedure was correct.

Cases I have met with have been few and of the more simple form of intracranial disease of otitic origin, and some of them are open to question, but I am convinced that the suggestion of operating early, is the safer to follow, and I have reason to believe that had I not waited so long in my first series of cases, and had my technique been better, the results would have been different.

To operate on a patient who will certainly die is a discredit to otology, and prevents or delays consent in other operable curable cases. To operate on a child whose symptoms are due, not to the extension of its ear disease, but to the toxemia of an acute infection, is to make a grave mistake. To fail to operate in a case of operable, curable mastoid or middle ear disease which is producing grave symptoms, is a fearful error. (Jackson.)

To all of which I agree, but in this age, what excuse can there be for allowing a patient, except in rare instances, to become hopeless before giving him the chance that only surgery can give?

I believe many of the hopeless cases and fatal results are due to delay and halting between two opinions. In these cases, everything is to be gained and nothing to be lost by the operation, and if the end is hastened by a few hours, and made easier by the operation, both relatives and attendants have the consolation of the knowledge that all was done that could be. A forward step will be taken when our patients are taught this, and also the wisdom of an autopsy whenever there is a reasonable request made. It is only in this way that the people and the profession are made safer than they are now.

It must be remembered that there is such a difference in the contour and structure of the epitympanum and the mastoid as to justify the assertion that there is no bony structure in the human body which presents so many deviations from an arbitrary standard type as the normal epitympanum and mastoid process of the temporal bone. The same multiplicity of variations is found on the inner walls separating the mastoid from the cranial cavities.

It is also true that in intracranial complications of otitic origin the definite symptoms of their existence unfortunately are either conspicuous by their absence or fallacious as to their significance, while the general condition of the patient may be such that the main localizing symptoms are incapable of demonstration. Especially is

this true in cases of cerebellar or cerebral abscess. An exact diagnosis as to the location is often impossible, and more than one operation may be necessary before the desired results are attained. In epidural abscess there frequently are no general symptoms of meningitis, nature walling off the area of meningeal inflammation from the cranial cavity, and often thus effecting a spontaneous recovery.

In the cases of extradural abscess, all but three were epitympanic, the others were in the posterior fossa, and were the only ones giving evidence of threatening serious brain trouble before the operation, these being of size and virulence sufficient to produce pressure and toxemia. In the former cases, there was no destruction of the bony walls bordering on the dura, the infection having taken place through a fistulous perforation in the wall. Had the bony roof of the tympanic and antral cavities been destroyed by the pathologic process, pus could not have collected between the dura and the bone.

Of course the more marked the symptoms of intracranial complications, the more urgent is the indication for operation. My own observations justify the conclusion, however, in cases of otitic diseases, whether acute or chronic, that symptoms of meningeal irritation are indications for immediate operation. Such symptoms are characterized by headache, photophobia, pain over the entire temporal bone, especially above the back of the ear, whether there be evidence of mastoid disease or brain pressure, with drooping eyelids, furred tongue, and loss of appetite, slow pulse and sluggish pupils. Other brain symptoms, such as somnolence, dizziness, and vomiting, optic neuritis and nystagmus may occur associated with an extradural abscess, yet the appearance of these symptoms is not to be relied on for making a differential diagnosis.

Operative procedure need not subject the patient to greater dangers than already exist, and therefore can be undertaken before the advanced stages of meningitis have already set in. As in threatened serious abdominal disease, appendicitis and peritonitis, immediate attempts are made to eliminate the primary focus of infection, so in the treatment of otitic meningitis, our aim should be to eradicate the primary suppuration in the temporal bone.

Only recently has otology received the full recognition it deserves, and the general practitioner who holds in his keeping the present and future health and hearing of his patients is just beginning to awaken to his responsibility—of warning them of the necessity of the most careful and scientific treatment in the nasal and accessory

sinuses. When this branch of the medical profession is aroused, we will have fewer cases of deafness and intracranial disease.

The one fact that now confronts parents, friends and medical attendants, is that the enemy is on them, and no enemy of the human body strikes with greater force and horror than the mention of meningitis or brain disease. The question now is not so much what phase of intracranial disease we have to contend with, but what shall we do? We are assured of the fact that unless the condition that exists is speedily brought to an end, the patient has everything to gain and nothing to lose by our most heroic efforts.

If the well known and accepted symptoms of the disease exist and there is evidence of suppuration in the ear, or history of previous suppuration, with vertigo, the two things to be done promptly and thoroughly are: First, to start vigorous elimination by purgatives, diuretics and diaphoretics, and these failing to relieve in a few hours, second, to drain the middle fossa through the tegmen tympani and antra after doing a radical exenteration of the tympanic cavity and the mastoid cells.

We lose valuable time and handicap our profession by waiting for differential points of diagnosis. Undoubtedly intestinal toxemia begins early in ear disease, and I know of nothing that so completely interferes with peristalsis resulting in obstinate constipation, as does pain in the ear. Here the vicious circle is started, in which we have first constipation, followed by faulty and perverted intestinal metabolism, disordered innervation, the urine being loaded with indican, with the result that marked evidence is shown in the toxic condition of the patient. This is no time or occasion for small doses frequently repeated or exploratory punctures; something radical is demanded from both a therapeutic and surgical standpoint. This is an occasion when in the very beginning we must hasten the results of our efforts, especially from the therapeutic standpoint.

My rule is to start active purgation with one large dose of calomel, five grains for a child under five years of age, and ten to twenty grains for those older. Give the full dose not only for its purgative, but for its antiseptic and sedative effect, to be followed in six hours by one ounce of castor oil, which is to be followed two hours later by one ounce of sulphate of magnesia. If the results of these do not indicate that the alimentary canal is empty and clean, a colon flush is given.

No nourishment is given but milk or broth every three or four hours, and not then if objected to by the patient. This treatment will assist materially in determining whether we are dealing with a meningeal irritation due to toxemia from the alimentary canal, or whether the chief source of trouble is elsewhere. In either event, it is the best preparation for any operative procedure that is to follow. If the meningeal symptoms are unmistakable, I do not think it wise to wait for the effect of drugs on the eliminative organs, but give the calomel just before starting the anesthetic, and hasten its action as soon afterward as possible.

In several instances where the patient was unconscious and in convulsions, the calomel was given after placing the patient on the operating table, the preparation for the operation being done during the administration of the anesthetic. In five cases where the symptoms of meningeal irritation and brain pressure were most marked and the cases seemed hopeless, as far as any treatment was concerned, this procedure of free elimination, drainage, and irrigation resulted in the recovery of three out of five.

In none of my cases were the meningeal symptoms increased by the operation, and I am convinced that early operation is less dangerous than waiting for exact diagnosis. McEwen's record of nearly eighty per cent of recoveries in cases operated upon for intracranial lesions, stands unparalleled. He, with Jansen, Herman, Heine, Ballance, Koerner, Lane and other well known Europeans, operates at the first extension of the infection of the intracranial cavity.

I know of no branch of surgery in which adequate hospital equipment, with trained assistants who are familiar with the technique of the operator counts for more, than in surgery of the cranial cavity. It is here that the "team work" and "system" so prominently brought out by the Mayo brothers are of paramount value. It is here that the anesthetist, operator, assistant and nurses are to understand thoroughly, and do their parts without the loss of time or friction. The minimum amount of anesthetic is to be used, and in certain parts of the operation, practically none is given; the minimum amount of laceration and bruising of the soft parts, concussion and jarring done away with by substituting Kerrison's forceps and rongeurs for the chisel and mallet.

The use of hot physiologic saline solution of a temperature of 115° to 120° F. from a fountain syringe, keeps the field of opera-

tion clean, controls hemorrhage, and keeps the dura warm and moist, as advocated by Sir Victor Horsley; the careful sponging with gauze wrung out of hot sterile saline solution, to avoid bruising the delicate structures.

In a word success largely depends on the thoroughness with which the operation is done, with the minimum amount of anesthetic, traumatism and shock, and the most careful sustaining of the eliminative and recuperative powers of the patient. In these cases an excess of indican in the urine is more to be feared than the presence of small quantities of albumen or sugar, so far as the ultimate results are concerned, and more is accomplished in the first twenty-four hours by giving physiologic saline solution by rectum or by hyperdermoclysis, than by giving milk, broth or any other form of nourishment.

In conclusion, I would urge that the keynote of the otologist dealing with intracranial lesions be: Operate early; and I believe that the otologist is the one to do the intracranial surgery resulting from infection of the ears.

Starr says, "It is an unfortunate fact that less attention has been paid to brain surgery in this country than it merited." Whether this statement is true, I am not prepared to say. If it is, it is a reproach to American surgeons, but not so great a one as is waiting for external evidence of pus formation in the mastoid or alarming evidence of septic absorption or intracranial infection before operating.

MEETING OF EASTERN SECTION, HELD IN BOSTON, JANUARY 4, 1908.

PAPER:

COMPLETE OCCLUSION OF BOTH ANTERIOR NARES.

ROBT. H. CRAIG, M. D.

When our worthy Chairman suggested that I should read a paper at this meeting, I felt honored, but rather diffident about acting upon his suggestion, as I have nothing of marked interest to bring before you; however, in looking over the records of cases that have come under my observation for some time past, I have selected three, which I trust will prove interesting, on account of their comparative rarity.

Complete occlusion of both anterior nares. The patient was a child, female, aged two years and eight months, brought to me by her mother in November, 1906, who stated that her child had been unable to breathe through her nose for the past two years.

When the baby was three months old the mother first noticed that she breathed with difficulty through her nose; a local physician was then consulted, and attempted unsuccessfully to pass a small catheter through the baby's nostrils. According to the mother's statement, the baby could breathe freely through her nose when born.

There was no evidence of rickets or inherited syphilis; the organs, so far as could be ascertained, were normal.

Examination showed a small child, anemic, marked nervous temperament, mouth open, and facial expression presented all the characteristics of a mouth breather. The nasal bones were almost on a level with the face, and presented a pronounced saddle-backed appearance, even for a child so young.

Both anterior nares were covered by epidermis, the same color as the surrounding skin, and which had apparently grown from the muco-cutaneous junction of the nares. I advised operation;

the mother readily consented; patient was anesthetized, an incision was made through the center of each plate of skin, both inferior turbinals were found in contact with the septum, but not adherent. I enlarged the space between the septum and inferior turbinals with nasal forceps and introduced two silk rubber drainage tubes. The after treatment was nothing worthy of note, and consisted in changing the tubes at stated intervals for cleansing purposes. The tubes were permanently discarded six months after the operation.

The result has been gratifying; the child breathes freely through both nostrils, and since the establishment of nasal respiration its appearance and general health have greatly improved.

A considerable number of cases of occlusion of the posterior nares have been recorded, but a cursory review of the literature has failed to reveal a case similar to mine.

Although the mother stated that the child could breathe freely through the nose when born, I am inclined to doubt her statement, and regard the condition as congenital. One must not forget, however, the possibility of the stenoses being caused by traumatism, possibly caused by the physician who attempted unsuccessfully to pass a catheter into both nares.

A brief reference to the embryology of the formation of the nose may prove interesting at this point. Heisler states the external nose first acquires definite form from about the eighth week by the union of the distal ends of the lateral nasal processes with the nasal frontal process, the former producing the alae and the latter the bridge and tip of the nose. In the third month the organ is unduly flat and broad, but from this time on it gradually assumes its characteristic form. From the third month to the fifth, each external naris is closed by a gelatinous plug of epithelial cells, therefore I am disposed to conclude that in this case the patency of the external nares was not established even at birth.

PAPER:

HYPERKERATOSIS LINGUAE.

ROBT. H. CRAIG, M. D.

Hyperkeratosis Linguae (the hairy black tongue). Butlin states in his admirable book on the tongue:

"This is a condition which has excited a great deal of interest, although its clinical importance is very slight. It is produced by an overgrowth of the epidermis of the filiform papillae, but there is no production of true hairs. The color is black, but it may be sepia brown, or yellow, as described by Dinkler, or even blue (Mourek). The color has nothing to do with the overgrowth of the papillae, but is due to the organisms clinging to them."

"And whoever is familiar with the various colors which are produced by the agency of micro-organisms can not doubt the power of organisms on the surface of the tongue to produce the black or blue colors, whether by the presence of color bodies or by the production of a soluble stain coloring the epithelial cells."

Case No. 1. Consulted me about the middle of last January regarding the peculiar appearance of his tongue.

Examination revealed a dark brown, almost black, patch on the dorsal surface of the tongue, immediately in front of the circumvallate papillae. Interspersed through the colored area were many tendrils of what appeared to be black hairs about one-eighth of an inch in length, evidently elongated filiform papillae. The condition was unaccompanied by pain or other untoward symptoms.

The patient was under my observation for about a week, the area was cauterized with a weak solution of silver nitrate, and a cleansing mouth wash prescribed. The treatment resulted in but slight temporary improvement; I assured the patient, however, the condition was not dangerous, and he failed to report for further treatment.

The history of this case failed to reveal any constitutional dyscrasia which might account for the condition. The patient ad-

mitted he was a heavy smoker, which might have accentuated, or possibly even caused the peculiar and rare appearance of the condition described.

Case No. 2. Male, aged 27. Patient had recently recovered from an attack of acute pharyngitis when he presented himself for treatment at the ear and throat clinic of the Montreal General Hospital.

Examination revealed almost the identical condition described in case No. 1, with the exception that it was not so pronounced.

A cleansing mouth wash was prescribed, and the patient was instructed to rub the tongue with a small swab, saturated in a weak carbolic solution, with the result that in three days' time the improvement was so marked the discoloration had almost disappeared. In this case the surface of the tongue was scraped, and microscopic examination by the house surgeon revealed many cocci and bacteria.

To quote Butlin again: "No explanation can be given as to the origin of the overgrowth of the papillae, except as regards the area in which it generally occurs. The dorsum of the tongue in front of the circumvallate papillae is the least exposed to friction, and it is here, if any, fur tends to be present.

"But Curtis saw a patient in whom the growth occurred behind the circumvallate papillae, a part specially free from air. Generally a black tongue causes no symptoms, and is discovered by

chance. Vollmers' statement that in half his cases the patients were syphilitic is entirely opposed to all other accounts. The duration of the patch is variable, but ultimately it tends to disappear spontaneously.

"On the whole, the best treatment is to take off the patient's attention by assuring him the state of his tongue is a mere curiosity."

DISCUSSION.

DR. CHENERY: I had one case come to the Boston Dispensary with black tongue very much like this picture. He was syphilitic, and the treatment was simply the ordinary syphilitic mixed treatment and a mild mouth wash. He came out all right.

DR. LELAND: It has been my fortune to see at least one black tongue in the City Hospital. It was much more circumscribed than this one Dr. Craig has shown. It was very black, and had hairs half an inch in length. I found the man was an excessive pipe smoker, smoking very strong tobacco in an old pipe, and I thought that might have something to do with it. I think one of the authorities mentions that as an etiological factor in black tongue. It is an interesting fact that Edwin Booth suffered from a black tongue for a long time, and he was an excessive pipe smoker.

PAPER:

ANOMALOUS FOLDS IN THE NASO-PHARYNX.

J. PAYSON CLARK, M. D.

It has been my experience, recently, to see two cases which have presented such an unusual condition in the naso-pharynx as to lead me to think them of sufficient importance to report.

Case 1. H. S., a boy ten years of age, was referred to me in April, 1907, by Dr. George A. Craigin, as a mouth breather. There was a history of deafness, increasing during the preceding year, also frequent head colds for a longer period, and occasional sore throat. He had undergone an operation for adenoids when about six years old. Examination showed lower turbinates swollen, and small, unimportant spurs on the septum. The tonsils were small, but there was a cervical gland as large as a small bean under the left angle of the jaw. Both eardrums were dull and retracted and apparently somewhat thickened. Hearing was much diminished. As the patient was extremely gaggy, a view of the naso-pharynx could not be obtained, but a rapid exploration with the finger discovered the naso-pharynx apparently filled with adenoids. The boy was operated on the next day, Dr. D. Crosby Greene, Jr., assisting. The tonsils were removed with the tonsillotome and punch. Considerable cheesy material was found in the crypts, although the tonsils superficially gave no evidence of this condition. A rather diffuse, but not large, adenoid growth extending into the fossae of Rosenmueller was removed with curettes. After thorough curetting there could still be felt with the finger what appeared to be folds of adenoid tissue attached to each Eustachian prominence and extending up to the vault of the pharynx. This was a condition of things which I had never met before, and I was considerably puzzled to account for it. On feeling again with the finger it was evident that these folds had not the characteristic feel of adenoid tissue. They were firmer and had a smoother, more velvety surface. They were perfectly symmetrical, one being to the touch ex-

actly like the other. They were as if the mucous membrane on the front and back of the Eustachian prominence had been continued in a fold to the vault of the pharynx, and they hung down very much like a dewlap. I asked Dr. Greene to examine the case, and he agreed with me as to the probable nature of the folds and that they were not adenoid tissue. Then the questions came up, What relation had they to the deafness, and should they be removed? The latter question was decided in the negative for the time being, because it was felt that to attempt to tear them away with adenoid forceps or curettes was certainly not justifiable, and furthermore, that their presence might have no relation to the deafness. But if the deafness did not improve, then these folds could be removed at a subsequent operation with a sharp cutting punch or other instrument adapted for the purpose, which would cut cleanly without tearing the mucous membrane and causing cicatricial contractions, which might be still more detrimental to hearing. The patient made an uneventful recovery from the operation. For the first few days there was no noticeable improvement in hearing, but at the end of a week, when the throat had practically healed, the hearing was much improved, and after Politzerization became almost normal. Without going into particulars, in the next few weeks the hearing steadily improved, and at the last visit about a month after the operation, was perfectly normal. The eardrums showed a corresponding improvement in their appearance. Consequently the extraordinary folds have been left undisturbed.

About a month later what was my surprise to stumble on the same anomaly in an adult.

Case 2. Harry S., a Russian Jew, twenty-one years old, came to the throat department of the Massachusetts General Hospital, presenting an ordinary case of chronic rhinitis with acute exacerbation. On looking into the naso-pharynx, however, I was astonished to see symmetrical folds of tissue extending from the pharyngeal vault to each Eustachian prominence, suggesting two curtains drawn back to disclose the posterior nares. There was not the slightest suggestion of there having been any inflammatory process in the pharynx, and the patient had never had an operation on his throat. These folds were present without causing any symptoms. To the finger they had a similar feeling to that of the folds in the first case. A small piece was removed from the free border of one of the folds by means of a punch for microscopical examination. This was sub-

mitted to Dr. William F. Whitney, surgical pathologist of the hospital, who has made the following report: "The specimen received from you, numbered 'Throat Room 80766,' consisted of a small piece of tissue from the growth of the pharynx. Microscopical examination showed the central portion composed of loose connective tissue, the spaces of which were filled with serum. It was covered on the surface by a single layer of columnar epithelium, the surface was more or less folded, forming crypt-like recesses, and in the tissue between were accumulations of small round cells recalling follicles. The tissue is best described as lymphoid, similar to that of the pharynx." To the sight and touch these folds appeared to be normal pharyngeal mucous membrane.

It seemed to me that we had in these two cases a very unusual condition, but one that was probably of developmental origin. After an inquiry among some of my acquaintances who are familiar with this field, and a consultation of such work as seemed likely to contain some reference to this condition, if it had ever been observed, I was unable to find any account of a similar case. Chiari, Zarniko, Heymann and others mention small bands and cords stretching from the Eustachian prominences to the pharyngeal wall, and all of us have seen such bands not infrequently. But these bands and cords are not symmetrical, are comparatively very small, and do not in any way suggest a relationship to the folds which I am describing. In order to see if embryology could throw any light on this condition, I visited the Embryological Department of the Harvard Medical School. There I saw Prof. Frederic T. Lewis, to whom I described the condition. He could give me no offhand information, but said he would be glad to look the matter up. Several days later I received a letter from him from which the following are extracts: "Professor Minot offered no suggestion as to its embryological significance, and Professor Terry . . . would like to know more exactly as to its location, particularly its relation to the pharyngeal recess (fossa of Rosenmueller) before saying that he had seen nothing of the kind. If the fold is between the auditory tube and the recess, it is in the position of the arch which lies between the first two gill clefts of the embryo (counting the spiracular cleft as the first). If it has any broad significance I should expect to find a suggestion of a similar fold in animals, and it would be easy to look for it in sheep, pigs, cattle, rabbits, cats and others. If these show no indications of the fold it is probably an adventitious development of the mucous membrane."

The folds are situated between the auditory tube and the recess, and are consequently "in the position of the arch which lies between the first two gill clefts." What the significance of this situation may be, and whether or not that significance is a broad one, the writer must leave to the decision of the comparative anatomist, since he has not the opportunities or training for making the necessary investigations.

Whatever may be their anatomical or embryological significance, they are evidently of extremely rare occurrence in the human subject. Clinically they appear to have no great importance, except that if they occur in conjunction with adenoid growths they may, by their bulk, increase the deleterious effect of the latter or may be mistaken for them, and an attempt to tear them away with adenoid forceps or curettes might do serious damage to the Eustachian tube and prominence. Before closing, I wish to express my thanks to Dr. W. F. Whitney and Dr. F. T. Lewis for their interest and assistance, and to Dr. D. A. Heffernan for looking up the literature.

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PAPER:

STRICTURES OF THE ESOPHAGUS DILATED THROUGH THE ESOPHAGOSCOPE. REPORT OF A CASE.

J. PAYSON CLARK, M. D.

Since esophagoscopy may be said to be still in its infancy, or, at most, early childhood, and only a comparatively few laryngologists have had any experience in its application, it seemed to the writer that the report of the following case might be of interest and value:

Arthur St. P. L., age six, of French Canadian parentage, was brought to the Throat Department of the Massachusetts General Hospital, on June 10, 1907, with the history of having swallowed a small quantity of caustic potash about six months before. For a few days after the mishap he could swallow nothing. Then he was able to swallow liquids, but not solids until three days before coming to the hospital, during which time he could not swallow liquids. On passing bougies a firm obstruction was encountered about eight inches from the incisor teeth. The patient was then etherized and a Jackson esophagoscope passed. This brought up in an apparently blind pouch or dilatation seven and a half inches from the incisor teeth. The greater distance shown by the bougie was doubtless due to its flexibility. After considerable search no outlet was discovered. After recovery from ether it was found that the patient could swallow liquids with apparent ease.

To facilitate the finding of the lumen of the stricture he was given, on the morning of the thirteenth, silk thread in a glass of water to swallow, the nurse keeping hold of one end of the thread which was then fastened to his cheek with a strip of plaster. He was then etherized and the esophagoscope introduced. The thread could be easily followed as the tube was pushed slowly down until the latter reached the dilatation above the stricture, when the silk thread could be seen disappearing through a minute opening. The stricture was too small to admit bougie No. 15 F. at first, but a No.

13 was passed and then a No. 15. During the next fortnight three or four unsuccessful attempts were made to pass bougies without the aid of the esophagoscope and general anesthesia.

On June 26 the boy was again etherized, and guided by the esophagoscope bougie No. 18 F. was easily passed, No. 20 with somewhat more resistance. It was discovered at this time that there was another stricture, of larger caliber than the first had been, about an inch beyond the first. This was also dilated. The use of the silk thread was found unnecessary at this second operation on account of the larger size of the lumen. On June 28 another unsuccessful attempt was made to pass bougies without ether and the tube. On July 1, bougies Nos. 18, 20 and 21 were passed through the esophagoscope under ether anesthesia. The patient could now swallow soft solids. On July 31, bougies up to and including No. 23 F. were passed under the same conditions. There was generally some bleeding when a tight-fitting bougie was used. Again, on August 28, bougies Nos. 20, 21 and 23 were passed in the same manner. The patient was then instructed to report once a month for treatment. Up to the last visit bougies could not be passed through the stricture without the tube. At last accounts the boy was eating without difficulty.

According to the table compiled by Mosher from Starck, the length of the esophagus from the teeth to the cardia in a child of five years of age is 26 cm. For a child of six it would probably be between 26 and 27 cm., or about ten and a half inches. This would bring the first stricture in this case about three inches from the cardia. Dr. Chevalier Jackson[1] says: "Flexible bougies of 8 mm. in diameter should pass in infants and children up to ten years in age." Bougie No. 23 F. is between 7 and 8 mm. in diameter. It is evident therefore that the strictures were dilated to approximately the normal caliber of the esophagus for a child of six. An interesting feature of this case was the marked dilatation of the esophagus above the stricture. This large pouch made the search for the very small lumen of the stricture a very long and tedious one. Jackson says: "Very often this (the lumen of the stricture) will be of almost pinhole size seen in the flat wall against which the tube mouth is gently pressed. Often prolonged exploration of a diverticulum, or a number of diverticula, will be necessary before the strictural opening is discovered." The previous swallowing by the patient of a silk thread, a well-known device connected with older methods of treatment of esophageal strictures, will obviate any prolonged ex-

ploration, the white thread forming a beautiful guide to the operator and saving much valuable time.

The reason for the inability in this case to pass flexible bougies even after the strictures had been considerably dilated, seemed to be the relation of the lumen of the stricture to the dilatation of the esophagus, the latter acting like a diverticulum in which the bougie stopped without being able to enter the stricture. This dilatation will doubtless slowly contract now that the condition of the esophagus below has become more normal.

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DISCUSSION.

Dr. Jackson: I would like to compliment Dr. Clark on the painstaking way he went about this case, and the very good results. These cases will try the patience of any one. Little children will go on through life struggling the best way they can, and making shift to get down enough food to sustain life.

By painstaking work a great deal can be accomplished. Dr. Clark brings out some very valuable points. The suggestion of swallowing the thread I consider very valuable. The occurrence of the diverticulum above the stricture is quite common, and when the ordinary bougie is passed blindly it usually ends up in that diverticulum. Many patients have gone to their grave from blind bouginage. Any attempt to find the lumen below the diverticulum simply perforates the bottom of that pouch. The diverticulum is always inflamed, occasionally it is ulcerated, and if you pass blindly a soft bougie without the aid of an endoscopic tube, you may find that ulcerated point where the esophageal wall is thin and go right through it.

I think the way Dr. Clark handled his case was exactly right. I do hope that every one will get up enthusiasm enough to take these poor wretches and do something for them.

PAPER:

REPORT OF A CASE OF TUBERCULOSIS OF THE TONSIL WITH REMARKS ON THE ADMINISTRATION OF POTASSIUM IODIDE IN TUBERCULOSIS.

NORTON L. WILSON, M. D.

Mr. L. H., age 46, a German, height five feet eight inches, weight 140 pounds, was an employee in the Singer Factory as a fitter of shafting. He was referred to me for diagnosis on the fifth day of April, 1906. For the year previous, he had had symptoms of tuberculosis of the lungs; and about three months before he came to me, he had what his doctor thought was an attack of tuberculosis of the bladder. At the time I first saw him, both of these conditions were improved, but he complained of severe pain when swallowing.

He had never had hemorrhages, and at the time of his call his temperature was 98 degrees. He felt chilly at times, and occasionally had sweats. There was no huskiness of voice, but he complained of pain in the throat, especially during deglutition. He had a slight cough, which was more pronounced in the morning.

Examination of the throat revealed the fact that the left tonsil was red and jagged and covered with a whitish yellow film, which, when removed, showed broken down tissue with superficial ulceration involving the crypts in the lower portion, while the upper portion of the tonsil showed well marked yellowish points, surrounding the crypts. An arc of red and yellowish white points, somewhat resembling mycosis, spread over the soft palate. The mucous membrane in the mouth and pharynx was pale as was also the larynx. No involvement of the larynx was detected. The maxillary gland on the left side was slightly enlarged. Examination of the chest showed a small cavity in the upper part of the left lung. The pathologist reported tubercle bacilli in the sputum and in scrapings taken from the tonsil.

My diagnosis was secondary tuberculosis of the tonsil involving the soft palate, and to me it seemed a clear case.

Not long after his consultation with me, he consulted another physician who diagnosed the case as one of syphilis, and in this he was confirmed by another doctor. The patient was put upon twenty grains of potassium iodide three times a day. Immediately the lungs began to break down, the throat symptoms became rapidly worse, and he died in agony early in July. I do not mean to say that this patient would not have died, but I do believe that his comfort was neglected and his death hastened by the course of treatment pursued. This I have observed in several similar cases, and I therefore plead in behalf of the tuberculous patient, that he should be spared the damaging effects of potassium iodide.

Tuberculosis of the upper air passages is of interest to the members of this society, not alone because we see more of it than the other members of the profession, but because we too are apt to fall into errors in making a clinical diagnosis. More than once have I seen the disease diagnosed as tuberculosis, and the patient make a rapid recovery after the liberal use of potassium iodide. On the other hand I have seen cases of tuberculosis diagnosed as syphilis and potassium iodide administered with most disastrous results.

Tuberculous patients do not tolerate iodides. It therefore behooves us to use extreme care in making the diagnosis, before we conclude to put them upon potassium iodide.

It is not often that tuberculosis of the tonsil is mistaken for malignant disease, but not infrequently it is mistaken for syphilis. We can not always rely upon the laboratory diagnosis. All of us have, without doubt, had eminent pathologists give us a report of tuberculosis, when tubercle bacilli were not found, but giant cells were present, and yet the patient recovered upon mixed treatment.

How then shall we know tuberculosis when we see it? First of all we should get as complete a history as possible. If there is given to us a history of syphilis, we naturally conclude that a patient's present condition is syphilitic; but, even under these circumstances, it is not safe for us to exclude tuberculosis for the two diseases may co-exist. If we are told the patient is living with a tuberculous individual, has a cough, fever, sweats and consolidation in the lung, it is safe for us to conclude that the trouble in the upper air passages is of a tubercular nature.

While I believe in primary infection of the upper air passages, I am convinced that many of the so-called primary infections are secondary in fact, and that a lung lesion, which really exists, has not been discovered. The typical symptoms are, however, frequently wanting, and we often have a hyperplasia of the tissues in the larynx or tonsil which is not always easy to diagnose.

After taking a complete history, it is my habit to look for tubercular lesions elsewhere, and not always to trust to my own judgment in such matters, but to have the lungs examined by an expert, and a tuberculin test given if necessary.

It goes without saying, that the sputum is always examined, and, if an ulcer is present, scrapings should be taken from this broken down tissue and submitted to the pathologist.

The local signs and symptoms should then be studied carefully, and here let me say that while enlarged glands in the neck and at the angle of the jaw are regarded, in some of the text books, as of extreme diagnostic value in syphilis, these enlargements may occur in acute primary tuberculosis or any other acute infection of the tonsil, mouth or nose. Fetor of the breath is also pointed out by some authors as being diagnostic of tuberculosis, but this is largely due to necrosis. The same odor may be found in any broken down tissue. A familiar illustration is to be found in the odor accompanying diphtheria. The character of the ulceration and the location of the hyperplasia are only helpful but not diagnostic.

In my opinion, the most important sign is pallor of the mucous membrane and the most important symptoms are cough and pain. The cough may be due either to the pulmonary affection, the condition of the pharynx or the irritability of the larynx. The pallor is the result of anemia, and may be seen in anemic individuals, but I have observed it more often than any other objective symptom. The pain is often complained of before ulcerations are present and is due to the infiltration of the tissues.

Iodide of potassium[1] and sodium are very diffusible and are rapidly excreted; setting free nascent oxygen and iodine at the points of elimination. They are remotely irritant to the mucous membrane. They induce great waste and rapid elimination of waste products, causing anemia, emaciation and depression. Mercury salts in small doses are blood tonics, improving the general condition, increasing the number of red corpuscles and body weight.

They soon, however, begin to promote waste by stimulating the lymphatic system.

A. Baurowicz[2] says, in doubtful cases of syphilis, if the iodide is not efficient, a mercurial treatment should be used in conjunction. The therapeutic test is the most reliable of diagnosis in many cases and should not be omitted before deciding upon the malignant nature of a suspicious growth.

I refer to the therapeutic test mentioned above because it is probably the course pursued by many of us, but if this same treatment is followed in a tuberculous case, the patient is sure to suffer from the effects of the experiment.

In cases of mixed infection of tuberculosis and syphilis, there is no definite rule regarding the administration of Potassium iodide.

Knopf[3] says "if the digestive organs are good and the iodide of potassium is well borne and does not produce coryza or increase the pulmonary catarrhal condition, it can safely be given in doses of from five to ten grains three times a day; but of course the patient has to be watched and the iodide must not be given for too long a time. If it is not well borne, interferes with digestion, or produces or increases the symptoms which are above indicated, mercurial friction should be resorted to."

Some syphilitics do better under potassium iodide, some under mercury, and some under mixed treatment.

It has been stated that some cases of tuberculosis have improved upon mercurial treatment and I am inclined to believe this is so if given for a short time only. Certainly it does not do the mischief that iodide of potassium produces and I therefore recommend that if we must resort to the therapeutic measures for a diagnosis, we should inject salicylate of mercury a few times and observe its effects.

Rarely should iodide of potassium be given to a tuberculous patient.

It has been my habit to suspend twelve grains of salicylate of mercury in one ounce of benzoinol and to inject thirty minims into the buttock once a week.

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3. In a personal letter to the writer.

PAPER:

A CASE OF PARALYSIS OF THE LEFT VOCAL CORD CAUSED BY PERITRACHEAL TUMOR.

W. E. CHENERY, M. D.

Without discussing the etiology of paralysis of the vocal cords, I wish to report this interesting case:

J. F., male, age 53, was first seen by me at the St. Elizabeth clinic, July 21st, 1907. He complained of hoarseness—no pain. He was born in Ireland and was by trade a plasterer. Family history was negative. Past history except for occasional colds very good. No history of syphilis or accident. He had been accustomed to hard work. Bowels regular and urine normal.

The present illness began one week before. He had been playing the flute for several hours and in imitating a bag-pipe, said he had "strained" his throat. The next day he could not speak above a whisper and had remained hoarse. He coughed occasionally with slight expectoration. There had been loss of weight. He was well developed but poorly nourished. No glands were felt anywhere. The nose and nasopharynx were negative. The color of the mucous membrane of the mouth was fairly normal.

Examination of the larynx showed the left cord motionless and in the cadaveric position. The left arytenoid did not move. All motion was on the right side. There was no evidence of ulceration or thickening anywhere. Color of cords nearly normal. The trachea was in the middle line. There was no dysphasia, no stridor, air seemed to enter bronchi well. Radial pulse synchronous. There was no evidence of central lesion.

Physical Examination. Heart—Apex beat in the 5th interspace. Sounds clear and regular. Pulse normal in rhythm and tension. No signs of aneurism of the aorta.

Lungs—Slight dullness at both apices to the 2nd rib and in both backs to angle of the scapula. Numerous fine râles, vocal fremitus increased.

Abdomen—negative; liver and spleen not palpable.

Extremities negative, reflexes present.

The probable cause of the paralysis was interesting. There was no history or evidence pointing to central lesion. Aneurism seemed to be ruled out by absence of signs. He had had no rheumatism, injury or disease except slight cough. The larynx did not show any evidence of tuberculosis. His age suggested pressure of the recurrent nerve from a new growth as to the probable cause, although its location could not be determined.

The patient then disappeared. Wishing to study the case further I sent for him at his home. He had been drinking and had developed an acute process in the lungs. He was sent to the hospital, where he died September 8th, 1907.

Six hours after death Dr. Timothy Leary made an autopsy and I append his report.

"On the left side of trachea lies a tumor mass 3x2x2 cm., arising apparently in the peri tracheal connective tissue and situated opposite the third to the tenth tracheal rings, inclusive, lying in part posteriorly—between trachea and oesophagus. New growth is a moderately firm mass and is closely attached to trachea. Opposite its mid-portion are seen two small flattened oval nodules projecting above the mucosa of trachea and corresponding in position to the sixth and seventh tracheal rings.

"New growth is penetrated below by the recurrent laryngeal nerve posteriorly, and anteriorly shows a second nerve trunk of considerable size, which may be the cardiac nerve of the sympathetic, a cardiac branch of the vagus, or possibly an aberrant cardiac branch of the recurrent laryngeal. The latter seems to be the most reasonable supposition, since the only nerves which can be identified as coming from the upper part of the tumor are the posterior thyroid branches of the recurrent laryngeal and, posteriorly, the main trunk, to the larynx.

MICROSCOPIC EXAMINATION.

"New growth consists of a branching epithelial meshwork imbedded in hyaline connective tissue. In the younger portions of tumor the strands of epithelial cells are broad, and the polymorphous cells

are well preserved. Mitotic figures are rare. In the older portions epithelial strands are slender, cells are fused, and concentric whorls of necrotic cells are abundant. The connective tissue in the more active part of growth is cellular, but shows a relatively large amount of hyaline intercellular substance with few fibrillae.

"In the older portions connective tissue shows few cells, which are vacuolated, and intercellular substance is wholly hyaline. In the central part of the growth large regions are neurotic.

"Tumor is extending into surrounding fat along perivascular lymphatics.

DIAGNOSIS.

"Peri-tracheal epithelioma.

"Death in this case was due to a bilateral pneumococcus pneumonia which was much more marked on the right side. On the left side process was limited to the base, consolidation was lax, and not homogeneous, some regions containing air. On the right side the process included a large part of lower lobe and consolidation was general.

"The left apex contained some obsolete tubercular foci.

"The other organs presented the usual picture seen in general septicemia, i. e., albuminous and fatty degeneration."

DISCUSSION.

Dr. Jackson: In many of these cases the radiograph will show a tumor such as that, and between the radiograph and tracheoscopy and esophagoscopy I think we can usually make an accurate diagnosis after the growth has reached any considerable size. Doubtless, if the patient had returned, and Dr. Chenery had had an opportunity to put a tracheoscope down into the trachea that tumor would have been seen. It has been my good fortune to have had an opportunity to make a diagnosis in several of these cases by either tracheoscopy or esophagoscopy showing a tumor of either the trachea or esophagus. This has occurred in patients who have had no difficulty in swallowing, pointing to the esophagus, and no difficulty in breathing, pointing to the trachea.

I have one patient at home now who has been paralyzed in both vocal cords for six years. Absolutely no lesion has been discovered, although I have examined him and a radiograph has been made. The neurologists can find no lesion, and certainly it is

time for something to have developed, if the case were an incident of tabes or disseminated sclerosis, or a central lesion. There are times when no man can make a diagnosis of the cause of laryngeal paralysis.

Dr. Halsted: I simply wish to emphasize what Dr. Jackson has said. In these cases the tracheoscope or laryngoscope, I think probably, would have shown those projections in the trachea.

In the past three weeks with an eight months' old baby, I was able to see distinctly and remove a fish bone one inch below the larynx in the trachea, and I feel quite sure that these projections in the trachea might have been seen by direct laryngoscopy or tracheoscopy, and a piece removed for examination.

Dr. Raymond: We all know that in the early stages of paralysis only the efferent fibres are affected. It is just possible that before only the efferent fibres were affected, and the increased congestion made the paralysis complete.

Dr. Richards: In referring to the history of the case, how did the hoarseness come on, at once, or from some break in the tumor? Did it come on gradually and the patient only notice it all at once? Did it produce the paralysis at once, or did it not?

Dr. Chenery: All I can say was that he had been playing a wind instrument for several hours, and then by some method had been imitating the bagpipe, so that he had strained his throat tremendously, and this thing came on directly afterwards. That is all the history I could get, and in asking others I learned it is possible for a condition like this to come on suddenly.

PAPER:

REPORT OF A FATAL OPERATIVE CASE SHOWING DEVELOPMENTAL ABSENCE OF THE OUTER SPHENOIDAL WALL, AND IN ITS PLACE A LARGE VEIN COMMUNICATING DIRECTLY WITH THE CAVERNOUS SINUS. AUTOPSY.

F. P. EMERSON, M. D.

June 28th, 1904. Eva H. S. L., 60 Blank St., Dor. Referred for treatment by Dr. B. Born, Mass.; 21 years; occupation, school teacher.

Past History—Catarrh since childhood. After eating frequently gags and vomits, which releases thick tenacious mass from the posterior nares. When vomiting does not occur, the act of eating excites hawking so that she has to leave the table. To avoid this she has of late used an anterior douche previous to each meal. There is pain over the bridge of the nose. Diphtheria at five years of age; severe attack. Light attack of measles.

Examination—Ant. nares normal—pharyngeal vault shows a tenacious mass covering its entire surface. Pharynx is glazed and dry. A bullet pointed probe shows a carious area in B. Sphenoidalis. General condition good.

July 5th—R. Ant. sphenoidal wall removed with Gruenwald forceps, together with Ant. end R. Mid. turbinate.

April 15th, '05—L. Ant. sphenoidal wall removed, together with Ant. end L. Mid. turbinate. Carious area on floor and inner wall curetted.

March 24, 1906.—No symptoms except some dryness of the pharynx.

November 17, 1906—Came back complaining of a return of former symptoms less marked. A large cell was found containing pus in the remains of the L. Mid. Turb., which was opened and drained.

May 25th, 1907—A thin tenacious grayish coating was found still present over the pharyngeal vault. Patient said that this still troubled her about twice a week. A thorough examination was again made, and in the R. sphenoid a carious area was felt on the outer walls with a soft mass posteriorly. This was taken to be polypoid, and but little force was used in probing for fear of exciting hemorrhage. The middle turbinate and the anterior wall of the sphenoid had already been removed. A curette was introduced and turned toward the outer wall, and on the withdrawal there was a profuse venous hemorrhage and a convulsion. The patient was immediately unconscious. A rapid tampon controlled the hemorrhage externally, but symptoms of brain pressure supervened, and she was removed to the Charlesgate Hospital, where she died at 11 o'clock, seven hours after the accident.

Autopsy by Dr. Leary, of the Tufts Medical School, in the presance of Dr. Arnold, Courtsey, Swan and others. A hemorrhagic clot filled the calvarium. A large varicose vein was found to occupy a dehiscence in the outer wall of the R. sphenoid, which communicated directly with the cavernous sinus and was the cause of death. The remaining ethmoid cells were everywhere necrotic, and about one-half ounce of muco-purulent secretion was found.

DISCUSSION.

Dr. Hinkel: I have had two serious hemorrhages after opening the anterior wall of the sphenoid, arterial in origin. The first, six or seven years ago, came on about ten days after operation. It yielded to tamponing, but broke out again the fifth day; after the second tamponing it ceased. There was for a moment a spurting hemorrhage immediately at the time of the operation; under a tampon it ceased. I had opened the wall downward and forward and the hemorrhage was arterial.

Two years ago I had another case in which there was an arterial hemorrhage; this occurred in my office, and I tamponed it and took the patient to the hospital, some quarter of a mile away. On the way there the tamponade worked loose and I re-tampened the nose at the hospital.

I allowed the patient to return to his home in Pennsylvania in about ten days. A week after he had reached his home another hemorrhage occurred. This was not checked until the carotid of that side was ligated. Before he left for his home I could see a

pulsating point toward the upper and outer wall of the face of the sphenoid, showing that there was a loss of the bony covering to this artery, but I did not anticipate the serious result that followed.

In both of these cases the hemorrhage was arterial, and I think the cause was probably a wound of a branch of the spheno-palatine artery.

I had a patient twelve years ago presenting the same conditions, in which death followed. The dyspnea was not relieved by tracheotomy, and the child died within a few days after operation. For various reasons there was not a post-mortem.

DR. CROCKETT: It has always seemed to me that the risk of curetting the sphenoid was so great a one that the possible advantage to the patient would not justify it. I think that the general consensus of opinion is that after you have secured an opening to the sphenoid you should let nature take care of the rest of it.

DR. RICHARDS: The type of instrument to use on the sphenoid would be one which grasps the anterior inferior wall between the invaginating blades, and absolutely cuts that piece out, bit by bit, without any chance of fracture to the side walls. I have used that type of instrument so far with success. The cutting must be in a forward direction and inferiorly. It seems to me that that type of instrument can be used with safety.

DR. COX: Is it the general consensus of opinion here that it is not a proper procedure, or, at least, that it is a procedure accompanied with an unjustifiable risk, to do any curetting in the sphenoid cavity? Should we content ourselves with enlarging the natural opening?

DR. EMERSON: For the last two or three years I have found that the sphenoid drains itself if you break down and enlarge the natural opening, and I have stopped curetting.

I do not wish to go on record as recommending the curette in the sphenoid, except to enlarge the opening. The instrument that Dr. Richards has spoken of has always been the one that I have used. I supposed that I was simply behind this polypoid mass, which I was using my curette to remove, and not curetting the outer wall. The extreme thinness of the outer wall shows how easily it can be fractured with any instrument.

PAPER:

THE COCHLEA AND ITS GENERALIZED TONE IMPRESSIONABILITY.

W. SOHIER BRYANT, A. M., M. D.

Tone transmission from the labyrinthine fluid to the cochlear nerve endings is accomplished without the intervention of any special resonating or tone selecting mechanism.

Post-mortem findings combined with ante-mortem functional tests show that there is no special part of the scala media of the cochlea which has a tone selecting function, and that therefore the tone perceiving mechanism must be located nearer the auditory center than the scala media.

CASES.

An exhaustive search of the literature has unearthed records of 26 ears which had had a functional test, autopsy and histological examination with sufficient data to warrant their consideration in this connection. The findings in two of these ears showed that impairment of the nerve supply of one of the extremities of the organ of Corti was not incompatible with normal tone perception, and that extensive pathological changes may be associated with comparatively slight loss of tone perception, or vice versa. These facts cannot be explained on the Helmholz piano-string theory.

I. (*3-1), and

II. (*3-2), same patient, and

III. (*3-3). In each all tones clearly heard. All three had degeneration of the ganglion in the basal whorl of the cochlea. According to the "piano-string" theory there should have been a loss of high tone perception in all these cases.

IV. (*1-b, left ear.) Loss of high and low tones. There was degeneration of the nerves in the first cochlear whorl. In the second and third whorls there was partial atrophy here and there. In

*Refers to the number in Bibliography.

this case the findings in the cochlea do not account for the loss of low tones on the piano-string theory. The middle ear was found practically normal.

V. (*2.) Right ear. Only fork C. 132 V. heard by air. Atrophy of the nerve fibers in the lamina spiralis ossia of the first whorl of the cochlea and degeneration of all the hair cells except the outer ones. In the second convolution most of the hair cells appeared normal. The third convolution showed no pathological change. The mucous membrane of the tympanic cavity and ossicles, normal, malleo-incudal and incudo-stapedial joints movable, membrana tympani and manubrium mallei normal. Foot plate of stapes not readily movable. In this case the findings did not account, by the piano string theory, for the great loss of tone perception, especially for the low tones.

VI. (*3-4), and

VII. (*3-5.) Two ears of the same patient. Low tones not heard. High tones perceived. Autopsy showed bands on the stapes and the round window covered over. A decrease in the number of ganglion cells in the basal whorl was found, contrary to expectations, based on the Helmholz theory.

VIII. (*3-6), and

IX. (*3-7.) Both high and low tones not heard. Autopsy: Oval and round windows full of connective tissue. Ganglion cells of basal whorl decreased in number. Pigment in cochlea. Loss of low tones not accounted for by the old theory.

X. (*3-11.) C^2, C, C^8 not heard in left ear. C of the intervening octaves heard. Autopsy: Fixation of stapes, connective tissue in round window, recent otitis interna, fistula of semi-circular canal. Ephithelial structures of cochlea destroyed, except membrane of Corti and the rods. The loss of tone perception would have been more extensive if special parts of the cochlea served for the perception of special tones.

XI. (*3-13.) Right ear did not hear, C^2-C^1, while the higher notes were heard. C of each octave, including C^8, were heard. Sequestra of two whorls of the cochlea, a portion of the osseus labyrinth and part of the stapes were found. In this case there was a wider range of tones than could be accounted for on the theory of sympathetic vibrations.

XII. (*4. Left ear.) Impaired hearings after typhus abdominalis. Tone limits, C^2-G^4. Autopsy showed sclerosis of the acoustic nerve. Number and thickness of ganglion cells in canalis ganglio-

naris greatly reduced, especially in the basal whorl. The old hypothesis could not account for the preservation of the middle register only in this case.

XIII. (*4-2. Right ear.) Right ear low limit G, and high limit Galton 3.5.

XIV. (*4-2. Left ear.) Left ear, low limit A^1, and high limit Galton 10.9. Autopsy: Both ears showed atrophy of cochleal nerve endings in the basal spirals of the cochleas. Organ of Corti well preserved. Fatty degeneration of the trunk of the eighth nerve. The Helmholz hypothesis can not account for the retention of the middle register in this case.

XV. (*5, case of S. J.) Left ear could hear tuning fork with difficulty. A sequestrum of the upper two whorls of the cochlea was discharged, followed by improvement in hearing, so that the patient could easily hear tuning forks of different pitch. (The other ear was unchanged.) The tone perception was too extreme after sequestration to be accounted for on the Helmholz theory.

XVI. (*6, case 1.) Vestibular symptoms; almost complete deafness for speech. Low tone limit C^1, high tone limit normal. Bone conduction shortened three seconds. Autopsy: Inflammation of entire vestibular apparatus. Organ of Corti almost intact; acoustic nerve free from degeneration. Acute middle ear inflammation; ligamentum annulare in part completely destroyed; acute otitis interna and purulent meningitis. The ganglion cells of the cochlea ganglion for the most part destroyed. According to the Helmholz theory there should have been a greater loss of tone perception.

XVII. (*6, case 2.) Acute right middle ear disease. Vestibular symptoms. Tuning forks all heard on both sides. Autopsy: Whole membranous portion of cochlea and canal destroyed. Purulent meningitis. The old theory cannot account for the preservation of tone perception in this case.

XVIII. (*6, case 3.) Fracture of base on right side; impaired hearing in right ear; left, normal. Low and upper tone limits, right, C^2 and C^5. Death from purulent meningitis. Autopsy: Right ear, acute middle ear inflammation; fissure of mesial tympanic wall extending across the cochlea. Organ of Corti in lower whorl destroyed, but in fairly good condition in upper whorl; inflammation of vestibular apparatus. The Helmholz theory would expect more lesions in the upper whorl of the cochlea.

XIX. (*7, case 1, right ear), and

XX. (*7, case 1, left ear.) Both ears, low tones from C^1 down perceived better than high tones. Autopsy: Chronic catarrh of middle ear on both sides. Striae acousticae hardly developed at all. Acoustic nerve normal. In both sacculi and utriculi there was a large number of black calcareous concretions. There is no explanation for this case on the Helmholz hypothesis.

XXI. (*8.) Cochlea was removed, after which all tones on the piano could still be heard, and hearing for the watch was improved. Not explicable on the piano-string theory.

XXII. (*9, author's case.) Removal of two-thirds of the cochlea. Patient could still hear fork C through bone conduction, as well as other forks of different pitch. These could also be heard through air conduction when resonator was used. Not explicable on the old theory.

XXIII. (*1, a, right ear), and

XXIV. (*1, a, left ear.) Both ears appeared at first glance to be confirmatory of Helmholz's piano-string theory. There was a loss of low tones and of high tones, with degeneration of the nerves of the first convolution of the cochlea. But when the details of the case are analyzed, it is apparent that the condition of the middle ear might account for the loss of high notes, as well as for the loss of low notes.

XXV. (*10, case 1.) Loss of hearing for upper end of scale. Autopsy: Middle ear normal; cochlear nerve in the mediolus abnormally thin; atrophy of spiral ganglion, especially in the first whorl of the cochlea; trunk of the acoustic nerve, also the endings of the cochlear and vestibular nerve, normal. The fact that the loss of the spiral ganglion cells and mediolar nerve fibers was not confined to the basal whorl would, according to the Helmholz theory, necessitate a partial loss of low tones, which was not the case.

XXVI. (*10, case 2.) Loss of hearing for high tones. Autopsy: Middle ear normal; trunk of acoustic nerve normal. Abnormally few cells in the spiral ganglion of the first convolution, also complete destruction of the organ of Corti in this convolution; in the other cochlear whorls the organ of Corti is still easily recognizable. The abnormal condition of Corti's organ in the apex whorls would suggest some impairment of low tone perception, according to Helmholz's theory.

Further anatomical proof of the inadequacy of the piano-string theory can be found in the author's article about to appear in the

Archives of Otology and Zeitsch. f. Ohrenheilkunde, showing that there is no known structure in the scala media which can act as a tone selector.

Conclusion. The records of functional tests followed by autopsy are lacking in detail, or else they prove that the various parts of the cochlea are not limited in their tone perceiving function, and perform this function indiscriminately for all tones.

Since written, the author has found another case similar to those cited in this article.

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*Refers to number of case considered in this text.

DISCUSSION.

Dr. Richards: I would like to know how Dr. Bryant explains the apparent fact that patients are sometimes deaf to certain notes in the scale and not to others, and sometimes hear certain words and not others. I had a patient two years ago who was almost totally deaf, certainly deaf as far as any conversation was concerned. I tried him now and then on certain scale notes; he would get some, then skip, then get some more, and so on. He had no hearing power for conversation, but would get a certain letter right now and then.

Dr. Bryant: I am sorry I cannot better answer Dr. Richards' pertinent question about tone gaps. I do not think these phenomena can be established upon definite pathological conditions. In my experience these symptoms are very uncertain, variable and unreliable. Often times a careful analysis of a case dispels the illusory tone gaps. Intense vibrations may be heard when weaker ones fail to be perceived. From the cases that I have had myself, and from published cases, I consider that whatever there may be of fact in tone gaps is based on middle ear or central disturbances. As you all know, the usual picture of loss of tone perception is one where the notes lacking are in unbroken series at one or both ends of the scale, a condition explained by my hypothesis. Normally the loss of perception of low tones is due to middle ear conduction anomalies, and loss of high tones to an impairment of the mechanism of the scala media.

PAPER:

PRIMARY MASTOIDITIS, WITH OBJECTIVE TINNITUS AURIUM.

S. MACCUEN SMITH, M. D.

It is with much hesitation that I use the term "primary mastoiditis," even in a case presenting such characteristic symptoms as that to which I am about to invite your attention.

It has always been my belief that primary mastoiditis, *per se*, never occurs. We all see, and not infrequently operate for the relief of, pathologic conditions of the mastoid process which give neither a history of trauma nor recognizable aural suppuration; nevertheless, careful examination will usually reveal some evidence of tympanic inflammation, which by continuity involves the mastoid process.

Cases that develop during a middle-ear disease, or occur after the same has run its course, cannot properly be designated as primary. It is conceivable, however, that bacterial infection, primarily attacking the naso-pharynx and tympanic cavity, may produce a temporary inflammation of the latter in its migratory passage to the mastoid process. My own belief is that the so-called primary cases are of this nature, the mucosa of the middle-ear cavity being in sufficient health to throw off or resist the virulence of the micro-organism. When, however, the bacteria have once gained entrance into the mastoid, the conditions existing there will prove most favorable to their rapid propagation and multiplication. In such cases all evidence of tympanic disease may disappear, for a time at least. The aditus becomes entirely occluded on account of the swollen mucous membrane, thus rendering the mastoid cavity practically a sealed cavern, possessing all the elements, such as darkness, heat and moisture, most favorable to the rapid development of bacteria, and effectually precluding their escape by the tympanic route.

In cases of this character, some days or even weeks may elapse before the adjacent tympanum or external auditory canal becomes

involved. In these cases, too, one of which it is my privilege to show you, there is an entire absence of external or middle-ear disease. The only symptom complained of in the case under observation was continued pain over the mastoid and objective tinnitus.

The only cases of mastoiditis to which we are justified in applying the term primary are those occurring by metastasis or resulting from trauma. Although hematogenous migration of bacteria into the mastoid bone cannot be proven, such a process by transmission through the blood and lymph channels must always be borne in mind as an important etiologic factor in cases of doubtful diagnosis. Moreover, no valid reason has ever been advanced to explain why bacteria cannot, by metastasis, be deposited within the organ of hearing quite as readily as an abscess formation in the liver or other viscus from a suppurating otitis, as a result of the same process.

From a study of patients seen by the writer, he has formed the opinion of cases in which the chief symptom is continuous, prolonged pain, that the diploe is first involved, the progress of the resultant osteo-myelitis slowly extending, externally involving the cortex and internally complicating the pneumatic cells. The inflammation, following the line of least resistance, as presented by the impaired internal plate, offers a plausible explanation for the extensive cell destruction and abscess formation seen in some cases.

The history of the patient which I present for you this evening is as follows: E. G., female, aged 17 years, consulted me at the Germantown Hospital in September, 1906, complaining of pain in both ears, the pain being more especially pronounced over the mastoid process. There was absolutely no evidence of disease of the middle ear, mebrana tympani, ossicles, or external auditory canal, although I could detect a passing redness reflected through the membrana tympani of the left ear. Had there been any inflammatory condition or thickening of the membrana tympani in either ear from some previous disturbance, it would, of course, have been impossible to observe this reflection through the drumhead. At first pain was elicited only on deep pressure over each mastoid process.

The patient was admitted to the hospital, that she might be kept under observation and receive proper treatment. The pain over the process gradually increased, and at the end of two weeks, when a sudden slight rise in temperature occurred, I operated on the left mastoid, notwithstanding the fact that the chief symptom still present was pain over the mastoid, which resisted all our efforts for its

relief, plus the recent temperature rise. There was no redness of the membrana tympani, and no inflammation of the external auditory canal or bulging of the posterior-superior wall of the same, and no impairment of hearing.

The patient was prepared for ether and a simple mastoid operation performed. After passing through the cortex, which was soft and unusually thin, a most extensive necrosis was found; indeed, caries of the entire cavity was complete. This would seem to be a case of primary mastoiditis, if we are justified in calling any case primary. Probably the infection traveled from the throat up through the Eustachian tube into the middle ear, and if such was the case, there was only a passing congestion of the middle ear, which promptly subsided without treatment, while the process in the mastoid increased. In this ear the entire tip of the mastoid was removed, but on account of the middle ear not being involved I did not interfere with the membrana tympani or the ossicles. The hearing in this ear was normal, and has remained so since the operation.

It did seem curious that a patient of good personal and family history, without apparent cause, should suffer so severely from pain in *both* mastoid processes, especially when all evidence of disease was absent except this persistent pain, which I was strongly persuaded at one time was only imaginary. As above stated, this patient was kept in the hospital for two weeks, during which period every line of treatment, including blood-letting, ice, etc., was applied, but without avail. When finally we decided to operate, I was still of the opinion that she was markedly neurotic, although in apparent good health, and that the best way to relieve her mental attitude was by a simple Wildes' incision. To my surprise, I found the cortex somewhat discolored, and showing points of bleeding. While this disclosure was embarrassing to the operator, it nevertheless demonstrated the exact condition beneath the surface.

Notwithstanding this experience, however, and apparently still disregarding the fact that she complained almost as much of the right mastoid, I felt that both ears might be benefited by the one operation. At all events, I concluded to wait for a day or two and watch results. As a matter of fact, the right mastoid did seem to improve—at least, this was the patient's impression, which was borne out by the temperature receding to normal—for a few days, but the pain became more severe, and at the end of two weeks the

temperature again rose to about 101° and the other mastoid was opened and proved to be in about the same condition as the left, with this exception, that at no time was I able to discover the slightest evidence of even a mild otitis media from repeated examinations of the ear.

From the very beginning this patient complained of a certain noise in both ears, of which, however, I knew nothing until some time after the second operation. She refrained from telling me anything about the tinnitus aurium, because she was afraid something else might be discovered that would necessitate her remaining longer in the hospital.

The noise resembled the ticking of a watch, and could be heard by the observer about twelve inches from either of the patient's ears, but could not be heard through her open mouth. So long as the patient was able to sustain a *full* inspiration the tinnitus absolutely ceased, both objectively and subjectively. At first the sound seemed to be synchronous with the heart's action, which caused us to suspect some circulatory disturbance as the chief etiologic factor in this particular phase of her case. Later, however, the ticking sound increased in frequency, so that it was one-third faster than the heart's beat. After the meeting of the American Medical Association held last June, I had the pleasure of showing this patient to a large number of members of the Section on Laryngology and Otology, at which time the pulse beat was 60, while that of the ticking tinnitus was 180. On October 29, or four months later, both showed a rate of 58 to the minute.

The noise had a distinct metallic sound, noticeable both to the patient and to the observer; this particular metallic note was lost however, when the diagnostic tube was used. Inflation by both Politzer's and Valsalva's methods showed the tubes to be patulous and gave a perfectly normal dry sound. During Valsalva's method of inflation the tinnitus ceased, just as when forced inspiration was practiced; this, however, may be explained on account of the mechanical action of both movements being similar.

This distressing tinnitus has continued uninterruptedly to date not having been modified in any manner by the mastoid operations and therefore presumably being entirely independent of said disease, although both began at the same time, and without apparent cause.

The tinnitus in this patient was involuntary, she not being able to exert the slightest control over the same, as has been reported in some cases.

A goodly number of objective tinnitus aurium cases have been reported, but as compared with subjective tinnitus it is quite a rare disease, and this is especially true in a patient having both ears thus afflicted, as well as having both mastoids involved. The question of etiology, therefore, becomes not only important, but most interesting. A number of theories have been advanced to explain this phenomenon, the one receiving most credence, and furthermore, that which may be said to be supported by the consensus of opinion, is that the noise is caused by a spasmodic contraction of the palatine or pharyngeal muscles, causing a sudden separation of the walls of the Eustachian tubes. If the noise in this case was caused by movements of the lips of the Eustachian tubes, I failed to notice the same on repeated rhinoscopic examination. In so far as could be noticed, the noise was unaccompanied by any movement of the drumhead or thyroid; was uninfluenced by mastication, deglutition, rapid respiration or by any movements of the head or body, or pressure over the carotids.

From the foregoing it is evident that the writer is unable to advance any satisfactory explanation of the actual cause of the ticking tinnitus in this case. However, he quite agrees with E. Vali,* who reports a case of objective tinnitus aurium, which he attributes to the clonic contractions of the fibers of the tensor veli palati muscle. He then gives it as his opinion that we do not hear the muscle contractions themselves, but rather that by their action the column of air undergoes movements, which cause the sounds. With this latter statement I am in hearty accord, for whichever muscle or set of muscles is responsible for the phenomenon, it is quite evident that the sound is dependent upon the disturbed air for its production, and not upon the simple mechanical action of a muscle. This theory would probably hold good for certain circulatory disturbances causing tinnitus aurium as well.

While examining the patient under consideration, I repeatedly found some slight muscular disturbance in the vicinity of the Eustachian tubes, although never enough, in my judgment, to cause the ticking tinnitus. However, the muscular contraction, as above stated, was present, and I may be justified, in the absence of a better explanation, in attributing the ticking tinnitus to spasmodic

*E. Vali, Archiv f. Ohrenh., Vol. 66, 1 and 2, p. 104.

muscular contraction near the Eustachian tubes, probably the upper fibers of the superior constrictor of the pharynx.

I requested Prof. Hobart A. Hare to examine the patient for the purpose of determining whether there might be some vascular condition that would account for this noise. Dr. Hare's report is as follows:

"I certainly have never seen a case like this before, and I cannot understand its mechanism, unless there be some vascular pressure upon the Eustachian tube which causes an approximation of its walls and then a separation of them, barring, of course, some difficulty in the external auditory canal, which I suppose you have eliminated. It would be interesting to find out what the effect of inflation with Politzers' bag would be — perhaps you have already tried this — and it is particularly interesting to note that if she takes a full breath and holds it, the sound stops, which would seem to indicate that it must be dependent upon blood pressure more or less directly. I do not find anything in her circulatory condition which gives a satisfactory explanation of her aural state."

Dr. Francis R. Packard also examined the patient, but was unable to account for this phenomenon.

PAPER:

A NEW FLAP IN THE RADICAL OPERATION.

BY GEORGE A. LELAND, M. D., BOSTON, MASS.

Some investigations I have been making for two or three years have resulted in the production of a flap for closing off the aditus from the mastoid cavity. Dr. Crockett has it down on the program as a new flap. I do not know whether it is a new flap or not, but I have not been able to find it in the literature. We have all probably had to go through the mastoid operation a second time. Then the patients kick, because they think it was not done properly the first time. I confess that the first secondary mastoid that I did I thought that the first operation had not been properly done; but after investigating a number, I concluded that it does not make so much difference how they are done first; whether they are properly cleaned out or not, they may be just as likely to need a secondary operation. These secondary operations come from a few weeks or months to a few years after the first. I have had occasion to operate during the last four or five weeks on four cases of secondary mastoid; some of the first operations I had done myself, some had been done by others. One recurred eleven years after the primary operation. In one of these cases I had operated on the child in infancy after scarlet fever. A year afterwards he was operated on for a secondary mastoid at the Carney, which was evidently done all right. Within the last two or three weeks I have had to operate again for a large amount of cholesteatoma which destroyed the whole cavity, and in that case the radical operation was done. The question arises, how can we stop the secondary infection of these mastoids? In these secondary mastoids, where we open up the old scar, we find there is a direct passage down into the middle ear through the aditus, lined with granulations in a fibrous envelope. Outside of this fibrous tissue there is more or less production of new bone. Now, if the aditus is not closed off there is no reason why this tract should not have a secondary infection, as well as the first one.

The middle ear is infected up through the tube, and then the infection goes up through the aditus and through this soft space leads to the outside surface and an abscess appears. In talking this matter over with some of the New York men, the desirability of shutting off the aditus, provided it was not shut off by the proper deposition of bone, was admitted. Dench and I thought that we would put a piece of sterile rubber tissue down over the aditus. I tried it several times, but of course it was not successful, because the middle ear side of the tissue was subject to infection, while the other was imbedded in granulations, so that these little pieces gradually floated out, and I had to go back to the old packing. On talking it over with Dr. E. H. Nichols, our surgical pathologist, I asked him if a piece of periosteum would live if put down over this aditus, separated from its attachment. Of course, that would not grow unless it were nourished, and he suggested that if I could get a large enough flap from the posterior surface of the mastoid it might grow if there were large enough attachment to give it nourishment. So for the last three years, after excavating acute cases, I have been taking a very large flap of periosteum from the bone back of the ear and packing the posterior end over the aditus, bending the bone surface forward to be applied to the posterior meatal wall. I may not live long enough to know whether these cases ever become infected again, especially if they are going to last eleven or more years before they have another infection, but theoretically and certainly in most cases this flap closing off the aditus will be sufficient to avoid further infection of the mastoid cavity. The periosteum forms a layer of bone, which closes off the aditus, and the posterior or skin surface grows fibrous tissue, which assists in filling the mastoid cavity. Nichols also said that if the flap were not long enough it would contract. I found that to be the case, and therefore have had to increase the length of the flap. The ordinary curvilinear incision is made through the skin and superficial muscles, so that we have a considerable thickness of tissue. This leaves more thickness between the fascia and the bone. Then the incision through the periosteum and overlying tissue is made from the mastoid tip upward and backward along its posterior border, then horizontally backward for half an inch or more, then upward to the temporal line, then forward on this line to the posterior mastoid limit, and then upward and forward through the temporal muscle, if necessary, to meet the first superficial incision. The idea is, we have got to take rather more periosteum than we might think

would be necessary. After the cavity is well excavated, this tip is put down over the aditus and packed firmly with small pieces of gauze or wicks. Then at the first dressing, four or five days after, provided the temperature has not risen, these wicks are not disturbed, but the rest of the cavity is unpacked and redressed. At the second dressing, three or four days later, on taking out the wicks, the flap will generally be found to be fastened over the aditus by firm adhesions. In some cases I did not get a large enough piece of periosteum, and at the second dressing I found that the periosteum had lifted up. I have one case here now where the periosteum had lifted up and did not cover the aditus; so I gave him ether, tore the periosteum loose where it had adhered to the posterior wall, and moved it over the hole, and now you will see that it has grown there, although it does not look so; but I have tried a probe and found it soft all the way down, with no passage into the aditus. If we have not obtained enough flap, and find that it does not cover the aditus, we can take away more or less of the posterior bony wall. We find at times that there is a good deal of tissue in the flap, making it quite thick, but this is an advantage, because it contributes to its nourishment, and also fills the cavity somewhat.

Now it occurred to me that if we could go still further and obliterate the posterior excavated cavity which results from the radical operation, we could save the patient a great deal of trouble from the treatment that is almost always necessary at intervals, for the removal of effete epithelium, wax, etc., that is so annoying. In these cases, after taking away the posterior wall, I have taken this flap and put it right down upon the facial ridge, pushing some of it down into the aditus, which of course is open, as well as the antrum, i. e., I shut off the aditus with the flap and have its edge fastened upon the facial ridge. In that way is made a new partition between the middle ear and the posterior cavity. The posterior canal wall is split, according to the method most desirable for the case in hand, and then the middle ear and canal are firmly packed, the anterior packing being made to fill out even with the facial ridge; then the periosteal flap is adjusted to the ridge and the aditus and anchored there by firm packing as in acute cases, and the posterior cavity is packed tight. At the first dressing, several days later, we take away the lighter packing behind, and do not disturb the packing on the periosteum. At the second dressing we can take this away carefully, not, however, disturbing the packing in the canal. About the third dressing we can take this out, and in most cases, so

far as I know (of course, I have not done it a great many times) the periosteum has adhered firmly in position upon the facial ridge, and the posterior cavity being then packed lightly, fills up very rapidly. Finally the middle ear is dermatized. I have not yet tried to dermatize it at the first operation, because I wish to be pretty sure that the flap was in good position. Only in one case have I had any difficulty, and in that case the lower part of the periosteum did not adhere, and there is a small cavity at the posterior inferior part of the wound. That, I think, was my first or second case. In a secondary operation I have had just lately the periosteum was almost useless, as it had a weak place in it at the middle, and the lower part sloughed out. This case proved to have an hereditary specific taint, and healed very poorly, so that the flap operation was a failure. One point I have learned to avoid in treating these chronic cases, and that is, placing the flap down there and expecting it to anchor itself and grow if the case is not a strong, vigorous and healthy one. If the healing is sluggish, the periosteum contracts and does not adhere, and therefore we have to go back to our old packing and dermatization just as before. If then the periosteum does not adhere we are not any worse off than before.

I have several cases here to show; one is a case of mastoid with hardly any pus in the cavity. I put the flap down, and it stayed, and the middle ear drained up through the external canal. The flap was not washed off or in any way compromised by the pus. In some of these cases which I have tried at the South Department, where the infection was extreme, there was a great deal of pus, and the necrosis was rapid. Here the flap always floated out. I have also tried it with the blood clot, as suggested by Sprague and seconded by Blake; but they were not successes. Now, if this firm fibrous tissue of the periosteum covers the aditus, the posterior cavity becomes a simple wound, to be filled with granulations, and the proliferation of the tissue from the posterior surface of the flap assists and hastens the process of healing very greatly. I also show two cases healed after radical operation, with no posterior cavity and the external canal only a little larger than normal; and five cases nearly healed after operation for acute mastoid, one double.

MEETING OF MIDDLE SECTION, HELD IN INDIANAPOLIS, FEBRUARY 22, 1908.

PAPER:

CAUSATION OF SUPPURATION OF THE TEMPORAL BONE.

WM. E. SAUER, M. D.

The vast majority of suppurations of the temporal bone are secondary to an inflammatory process in some part of the auditory apparatus. In discussing the causation of these suppurations, I will take up the etiology of acute middle ear suppurations, chronic middle ear suppurations, mastoiditis, and the suppurations of the labyrinth.

While it is admitted by some, that a purulent exudative process may exist without the presence of pyogenic bacteria, or at least without its being possible to demonstrate their presence, yet it may be safely stated, that practically all suppurative conditions of the temporal bone are due to the invasion and action of pathogenic bacteria. The bacteriology of the subject is to be taken up in the paper that is to follow. Therefore, I will deal only with the mode of invasion and the predisposing causes.

The invasion of the middle ear may take place through the Eustachian tube, through the external auditory canal, by way of the blood or lymph channels, by way of the labyrinth, and along the sheaths of the auditory and facial nerves, and from traumatism.

Invasion through the Eustachian tube is the most frequent. Infectious material may be carried by direct extension from continuity of tissue, or be forced into the tympanum by air or fluids. Inflammatory conditions of the nose and naso-pharynx frequently extend through the Eustachian tube into the middle ear by continuity of tissue. This extension of an inflammation or the in-

vasion of the middle ear is favored by the presence of nasal and naso-pharyngeal obstructions with their attendant condition of persistent congestion of the mucous lining of the tube and tympanum. These obstructions may not only interfere with the normal ventilation of the tympanic cavity, thus retarding or obstructing the venous circulation, with resultant exudation into the middle ear cavity. This exudation may become infected, as under these circumstances it offers a favorable medium for the development of pathogenic micro-organisms, which, according to Hasslauer, may be present even in the normal middle ear, or which may have gained entrance from the pharynx.

The infection of the middle ear by the air may take place when attempts are made to clear the nasal passages by forcibly "blowing the nose" when the nose or naso-pharynx contains infectious material. This may also occur through the employment of the Politzer method of inflation, or when the catheter is employed. Infection through the medium of fluids entering the middle ear through the Eustachian tube, occurs most often from the faulty methods in using the nasal and post-nasal douche; through the practice of snuffing up fluids into the nose, or while bathing some of the fluid may accidently enter the nose and mouth, and find its way into the Eustachian tube. This I have found to be a rather frequent cause of suppurative otitis media; and in most cases it was determined that the patient had visited one of the indoor swimming pools.

There are a number of acute suppurations of the middle ear which have their origin in the nasal cavities, which are the result of operative interference. During the period when the electro cautery was so indiscriminately employed in the nose and pharynx, acute middle ear suppurations were frequent sequels; and even today this may occur in spite of all precautions. Packing the nasal passages after operative procedures may also be the cause of an acute otitis media; especially if the tampon is allowed to remain in position for too long a time. I have known of a number of instances where an otitis media followed the removal of adenoids and tonsils.

Infection of the middle ear through the external auditory canal may occur as a result of solution of continuity of the drum membrane, either by traumatism or the action of an inflammatory process, or through the Rivinian segment. Traumatic perforations

the result of direct or indirect violence, with primary or secondary infection, are not infrequent sources of middle ear suppurations; otitis externa with its extension, according to Dench, through the Rivinian segment is also a cause of otitis media. More rarely the development of a vegetable fungus in the auditory canal may lead to a destruction of the drum membrane, with secondary involvement of the middle ear. The contact of cold water or cold air, if continued for a sufficient length of time, may cause an inflammation of the drum membrane with secondary middle ear involvement. Hot water or other irritating fluids poured into the external auditory canal, may produce the same result. Unskilled efforts at removing foreign bodies from the external auditory canal have resulted in injury to the drum membrane with infection of the middle ear.

Infection of the middle ear by way of the blood and lymph channels, may take place in diphtheria, measles, scarlet fever, tuberculosis, influenza and endocarditis. The infection of the middle ear through the labyrinth, or by way of the facial and auditory nerve occurs very rarely, but it may occur in cerebrospinal meningitis or from a meningitis from other causes.

Acute suppurations of the middle ear occur most often in children. The underlying factors accounting for the prevalence of the acute middle ear suppurations in children are, according to Kopetzky:

1. Constitutional diseases, and the hereditary influence of tuberculosis and syphilis.

2. The prevalence of the exanthemata during childhood.

3. Anatomic structures peculiar to childhood in the ear and its adnexa.

Among the general diseases given as predisposing causes are, rachitis, broncho-pneumonia, the gastro-intestinal derangements, or any disease which may weaken the body resistance. Kopetzky, with Koerner, holds that predisposing factors are tuberculosis or syphilis in the parent; the children themselves not necessarily being either tuberculous or syphilitic.

Primary tuberculosis of the middle ear is a comparatively rare affection. It is most often encountered as a primary disease in the mastoid. A secondary invasion of the middle ear through the Eustachian tube is not infrequent in older patients, and is seen most

frequently in patients with pulmonary tuberculosis; especially during the last stages.

Syphilis, according to Politzer, may also be the cause of a middle ear suppuration; the specific virus entering the middle ear through the Eustachian tube. It occurs most often as a consequence of a syphilitic naso-pharyngeal affection which directly involves the tube.

The exanthemata are among the most frequent causes of acute middle ear suppurations, and of these, scarlet fever is the most frequent and destructive in its effect. According to the views of the majority of investigators, the otitis is most often of a secondary nature, and the infection enters through the Eustachian tube. Though it is not impossible that the specific inflammation may involve the pharynx, tube and tympanum synchronously, in some cases the pharynx is not involved, showing that the infection enters by way of the blood or lymph channels. Levy describes a case of scarlatinal otitis which appeared seven days prior to the eruption. In spite of an early paracentesis the mastoid became involved and operative interference was necessary. The otitis media that occurs in the later stages of scarlet fever runs a much milder course. According to Bezold ½ per cent of all ear diseases are due to scarlet fever; and in from 20 to 30 per cent of all cases of scarlet fever there is an involvement of the middle ear.

Measles is considered as the next most frequent cause. The characteristic inflammatory macules (in the early stages of the disease), have been found on the tympanic mucous membrane, coincidently with their appearance in the mouth and naso-pharynx, and so are not secondary to an inflammatory condition of the naso-pharynx. Later in the disease, an infection may take place as the result of an inflammatory process in the naso-pharynx.

Diphtheria is also a frequent cause of middle ear suppurations. It rarely occurs as a primary affection of the middle ear. According to Hasslauer, there have been only three bona fide cases reported; namely, those by Krepuskas, Lewin, Gruenert and Schilling. The other cases occur as a secondary involvement through the Eustachian tube.

Influenza is also a frequent cause of acute suppurative otitis media. The infection may enter by way of the blood or lymph channels, and the otitis occur during the first or second day of the disease; or it may occur later, as the result of an infection enter-

ing through the Eustachian tube after the disease has practically run its course.

An acute suppurative otitis may also be the result of the direct extension of an inflamed parotid gland, as it occurs in mumps. This extension occurs through the glaserian fissure; or it may occur secondarily from the inflammatory affection of the pharynx and naso-pharynx, which sometimes occurs in this disease.

Acccording to Bezold, acute otitis media occurs in from 2 to 4 per cent of all cases of typhoid fever. The ear involvement occurs in the fourth or fifth week, and may be the result of a pharyngitis or of septic material being blown into the ear in an effort at clearing the nasal passages; or the infection may enter by way of the blood current from an endocarditis or from collections of pus in some other part of the body.

The anatomic structures peculiar to children, which, according to Kopetzky, are factors in tending to cause the prevalence of middle ear suppurations are: the adenoid tissue, the short and wide lumen of the Eustachian tube, and the embryonal character of the contents of the middle ear. He considers that the normal adenoid acts as a barrier to the entrance of particles of food into the Eustachian tube in infants. On the other hand, the effect of the diseased or hypertrophied pharyngeal tonsil, with its secretion of ropy mucus and resulting congestion of the pharynx, offers an attractive place for the development of pathogenic micro-organisms. Aside from the local effect of obstruction, their influence on the general economy by reducing its resistance, makes the presence of adenoids a most potent factor in the causation of middle ear suppurations.

All writers agree that the widely open lumen of the Eustachian tube, as well as its shortness and horizontal direction as present in children, favors the invasion of infectious material into the middle ear. The retrogressive changes that take place in the embryonal tissue in the middle ear of infants, also furnish, according to Weiss, Preizing, Kopetzky and others, soil for the development of pathogenic bacteria.

Chronic suppurative otitis media is usually regarded as a sequel to acute suppurative otitis media. An exception occurs in tuberculosis; as in this affection the process may appear with a chronic character from the beginning. Just when an acute case passes into a chronic one, is not easy to determine; but after a suppuration

has continued for two to three months, it is usually regarded as chronic. The general conditions that influence or cause the transition of an acute into a chronic case are, constitutional diseases which tend to lower the resistance of the patient, such as tuberculosis, syphilis, anemia and marasmus. The conditions which are important local causes of middle ear suppuration are, according to Politzer, 1. The granulations and polypoid growths of the mucous membrane of the tympanic cavity and on the membrana tympani, which develop in the acute stage. 2. The retention and causation of the purulent secretion in the lateral concavities of the tympanum, in the depression of the petrous bone, and in the cells of the mastoid process. 3. Caries which is developed in the mastoid or in another portion of the temporal bone during the acute course of the disease. 4. Chronic inflammations of the external ear, as is sometimes the case in erysipelas and chronic eczema. 5. Chronic blennorrhea of the mucous membrane of the naso-pharynx and ozena.

Just what part the micro-organisms play in the development of a chronic suppurative otitis is not definitely known, though an otitis occurring during the course of scarlet fever and diphtheria is more apt to be chronic.

There can be no doubt that a number of acute cases become chronic as a result of improper treatment and unhygienic surroundings.

Inflammations of the mastoid process may occur during the course of an acute or chronic middle ear suppuration; or they may be primary and the result of traumatisms. The inflammation of the mastoid occurring during the course of acute and chronic middle ear suppurations is due to insufficient drainage, either because of the opening of the drum membrane being inadequate for the accumulated discharge, or from a blocking off of the communication between the mastoid cells and the middle ear. If the views of Politzer are accepted, the mastoid process contains pus in every case of acute suppurative otitis media, and so long as the communication between the antrum and the mastoid cells is free, stagnation of the secretions in the mastoid does not take place, and no reactive symptoms are produced; however, it can be easily understood that if the opening is small, a swelling of the mucous lining of the cells alone may produce an occlusion with stagnation, and as a result, the osseous tissues may become involved. Other caus-

es which tend to mastoid involvement during middle ear suppurations are, exposures to wet and cold, the forcible injections of fluid into the ear, or the employment of the air douche. The experiments of Schutz on the cadaver have proven that the employment of the air douche in an acute otitis media when the perforation in the drum membrane is small, is a dangerous procedure, as some of the pus may be forced into the mastoid cells. In seventeen of the eighteen cases on which experiments were made, when the perforation in the drum membrane was small, the stained pus placed in the middle ear through this small opening was found in the remotest cells of the mastoid after catheter inflation, but he found the pus in only one of sixteen in which the same experiment was made in the presence of a large opening in the drum membrane. The occurrence of mastoid involvement is frequently seen in certain infectious diseases, as in scarlet fever, measles, diphtheria and influenza. The conditions which favor the extension of a chronic middle ear suppuration into the mastoid cells are, narrowing of the external canal, the presence of polypi and granulations, and the formation of cholesteatomatous masses, as these interfere with drainage and lead to caries and necrosis of the bone.

Primary inflammations of the mastoid are mostly the result of traumatisms. Tuberculosis is, according to Henrici, a comparatively frequent cause in children. He found that one case out of every five mastoid cases operated upon by him was tubercular; and he believes that the majority were primary. Ismer believes that 13 per cent of all mastoid cases in children are tubercular, and half of those are primary. Haike demonstrated that out of five cases of tuberculosis of the mastoid examined by him, three were primary.

It is possible that a number of so-called cases of primary mastoiditis are really secondary to a middle ear involvement, so slight as to be overlooked.

The invasion of the labyrinth may take place through the middle ear, by way of the blood and lymph channels, from the cranial cavity by way of the aqueducts, the facial and auditory nerves, and through trauma. The great majority of labyrinth suppurations result from the extension of middle ear suppurations. In the acute otitis media due to scarlet fever, measles, diphtheria and influenza, such extension is more apt to occur. In the chronic cases, tuberculosis and cholesteatoma play an active part. This exten-

sion may take place through the anastomosing vessels of the middle ear and labyrinth, or through a destruction of the bone from inflammatory processes. The labyrinth capsule may also be opened during operation on the middle ear, or penetrated by foreign bodies entering through the middle ear. According to Hinsberg, the most frequent paths of entrance for this infection are the round and oval windows, the promontory and the horizontal semi-circular canal.

The invasion of the labyrinth by way of the blood and lymph channels, as it occurs in mumps, and the invasion from the cranial cavity by way of the aqueducts and nerves, as well as in fractures of the temporal bone, are less important from a surgical point of view.

Regarding the frequency of labyrinth suppurations, Friedrich states that the labyrinth is involved in about 1 per cent of all the cases of middle ear suppurations; but as our knowledge of this subject increases, it will be found that the percentage of labyrinth suppurations will possibly be increased.

PAPER:

TREATMENT OF SUPPURATION OF THE TEMPORAL BONE OTHER THAN SURGICAL.

GEO. F. KEIPER, A. M., M. D.

That all cases of suppuration of the temporal bone, especially all cases of what we commonly know as mastoiditis, do not need either the simple or radical mastoid operation, goes without saying. In fact, many such cases are cured without such operation. But do not construe this to mean that because many are so cured, all ought to be. They won't.

At the outset let us understand that some of the cases which we see are in such condition that any attempt at other than immediate

operation is a reckless waste of time, both for physician and patient. But on seeing a case upon which, after careful examination, we decide not to resort to operation, at least for the present, the question arises, what shall we do?

We shall dismiss that very rare affection, primary periostitis of the temporal bone, and confine ourselves to the treatment of middle ear suppuration, whether acute or chronic, and mastoiditis.

Some general principles of procedure ought to be laid down to cover all these conditions:—

First—The patient demands a thorough examination, both locally and generally. A full history of the case will be necessary. This will determine the general condition. The general condition is of great importance, not only with reference to the cause of the disease, but also with reference to the extension of the disease to other tissues of the body distant from the seat of the primary focus. I have seen cases where suppuration in the temporal bone showed itself elsewhere. A recent case in St. Elizabeth Hospital, this city, had synovitis of the right knee joint, due to pus in the temporal bone.

Second—The physician must always keep in close touch with the patient.

Third—A blood count is of great value to determine, first of all, the number of white cells and the relative proportion of the polymorpho-nuclears; second, the number of red cells, and lastly, the percentage of hemoglobin. This should be made frequently during the progress of the disease.

Fourth—The microscope must be further used to determine the character of the pus, especially as to the bacteria present therein. For if we find pneumococci, we may feel encouraged to try abortive treatment. The figures of Cunningham, while house surgeon at the New York Eye and Ear Infirmary, show that 90 per cent of cases with pneumococci in the aural discharge recovered without operation, and a mixed infection without streptococci allowed 66 2-3 per cent of recoveries, without operation.

This is quite in contrast with pus showing streptococci; whereas only 14 per cent recovered without operation, and with pus showing staphylococci, there were only 33 per cent recoveries without

operation, while a mixed infection with streptococci, only 8 per cent recovered without operation. The rest required operation.

"Under pneumococcus," writes Dench, "are included those cases in which the infective organism was a diplococcus resembling closely the pneumococcus, but not of sufficiently definite character to state positively that they were true pneumococci."

In other words, the examination of the blood to determine the leucocytosis, and of the pus of the ear to determine its bacteriology are essential prerequisites that we may know approximately, if our case has a prospect for recovery with or without operation. The results of the examinations should be communicated, either to the patient, his or her family, or nearest friends or relatives.

Fifth—The pus should be examined for bone dust for that indicates caries.

In otitis media suppurativa acuta, the first essential is to secure good drainage through the external auditory canal. If the drum-head is not ruptured, the cause of good drainage will be best served by a free incision in the drum-head posterior to the malleus handle. This will not only relieve pain, but will prevent pus being dammed back into the mastoid antrum to produce necrosis by pressure. Again the nice clean incision will promptly heal, after the pus has ceased to flow, whereas, if nature, after intense suffering to the patient, ruptures the drum, an irregular opening will be produced, which may never heal, besides causing unnecessary deafness to the patient. Then drainage is best secured by the gauze wick carried right down to the perforation. To complete the dressing, the external ear is covered with an absorptive dressing in the shape of a pad of absorbent cotton. When the gauze wick is saturated with pus, it must be removed, the ear syringed and dried, and insufflated with boric acid powder. Then a new gauze wick is introduced, as was the first one. The pus in the middle ear should have been previously aspirated, not Politzerized.

If pain persists, the application of heat will be very agreeable, preferably by the Japanese pocket stove, which, when properly lighted, will maintain heat evenly for two hours, the degree of which may be regulated by the amount of wrapping around the instrument. This should be tied on the head over the ear. This is better and handier than the Leiter Coil, or the hot water bottle. Morphia should be used cautiously.

The best place for the patient is in bed, in a sunny, warm room, which is well ventilated and clean. The bowels should be opened after this fashion, if the patient be an adult:

℞ Hydrarg. Chlor. Mitis .....gr. ii
Quininæ Sulph .....gr. xviii
M. Ft. cap No. vi.

Sig.: One every three hours until the bowels move. If they do not move within two hours after the last one is taken, then a dose of sulphate of soda or sulphate of magnesium is indicated.

The above will be modified as to dosage according to the age of a child when used on a child.

Attention to the nose and naso-pharynx will also be necessary, and if the patient be suffering from acute rhinitis, the application once daily to the nasal mucous membrane of a solution of the following will be valuable:

℞ Iodin .....gr. v
Potass. Iodidi .....gr. xv
Glycerin .....oz i
M. (Gleason.)

The treatment of otitis media purulenta chronica will vary with the cause, which may be local or general, or both. If general, it may be due to anaemia, tuberculosis, scrofula, syphilis or la grippe. If local, the discharge may be due to abscess of the external auditory meatus, but most frequently to inflammation of the middle ear and mastoid cells, and less so to polypi.

Hence for constitutional treatment, iron will be indicated for the anaemic; potassium iodide in large doses, and mercury for the syphilitic; for the emaciated quinine and sodium salicylate; for the tuberculous, sunshine and fresh air; nourishing food, with cod liver oil and creosote, and for all, exercise in the open when possible.

For local treatment, the first thing is cleanliness, secured by judicious syringing with warm, sterile water, followed by careful drying of the parts. If polypi are present, remove; if granulations, use warm alcohol in the external auditory meatus. The warm alcohol is a good cleaning agent in any event. It may be forced into the middle ear by pressing the tragus into the canal completely filled with the alcohol. If the perforation be in Shrapnell's membrane, the syringing must be done with a delicate syringe point introduced through the perforation. Peroxide of

hydrogen should be carefully used, and used only on the applicator to soften and loosen crusts in the external canal. The drying of the parts may be secured by heated air. Powdered boric acid may be blown into the ear, but not in sufficient quantity to obstruct the canal. Richards recommends pyoktanin (one part in nine parts of boric acid), as an insufflating powder, and it seems in the writer's hands to do all claimed for it by its author, though it does discolor the ear and adjacent parts a deep purple, which is sometimes hard to remove even by alcohol.

Again we may use the gauze wick, as in acute suppuration, but with care, for many cases do better with the dry treatment with boric acid powder.

As to drops, the best remedy to use in the middle ear is nitrate of silver in solutions of varying strengths. D. A. Kuyk of Richmond recommends beginning with a six per cent solution, gradually increased to a 25 per cent solution, to be forced into the innermost recesses of the ear by the Siegel otoscope (Journal A. M. A., 1905.)

For acute mastoiditis, the first essential is free drainage. If the hole in the drum is not large enough, enlarge it by a Graefe cataract knife. If pain is present, relief will be most grateful. It will be best to aspirate the pus, instead of blowing it out by the Politzer method, though by Politzerization, the ear can hardly be more infected than it is. Free drainage will be promoted by the use of the gauze wick as above. The patient should be put to bed and kept there until symptoms have subsided. The writer is partial to the application of two Swedish leeches to the mastoid process, early in the disease. The writer has always preferred heat by means of the Japanese pocket stove. There has been some controversy as to the relative merits of heat and cold, as local applications in this disease; but to my mind, consideration of the Principles of Surgery, which are the principles of all surgery, local as well as general, settles that point. Cold certainly contracts dilated inflamed blood vessels, but while doing so, locks up in the tissues the bacteria of suppuration present, and does even more harm by reducing the vitality of the parts. The worst cases of abscesses of the brain seen and operated upon by the writer have been those where cold was used as a local application. Note further, that cold is advised to be used only twenty-four to forty-eight hours. What we want in these cases is not stasis, but diape-

desis, and as McGuire (*Virginia Medical Semi-Monthly*, Nov. 4, 1910) writes in his "Principles of Surgery," "heat acts by soothing the sensory nerves, thus diminishing pain; by drawing blood to the surface, thus acting as a derivative; by favoring of collateral circulation, thus relieving stasis and by liquefying exudation and hastening either its absorption or conversion into pus."

The preference is for the Japanese pocket stove applied day and night, until symptoms abate, or until no apparent improvement ensues. There is no indication for removing it after forty-eight hours, as in the case of cold.

Morphine is contra-indicated, because it masks the symptoms so that it becomes impossible to tell whether the patient has local tenderness. It produces a false security, which leads the patient to believe himself better when he may be worse. The heat will, in the great majority of cases relieve the pain and tenderness. For sleeplessness, chloral hydrate and bromide of potassium or sodium may be used.

Blisters are inadmissible, because they make careful examination of the mastoid process impossible, because the blistered surface feels painful when touched.

Of course these patients do better in bed in a well ventilated, clean room, bright and cheery from direct sunlight.

The bowels must be opened by the calomel purge outlined above. The treatment for the general condition will vary as to the cause, whether it be the exanthemata or la grippe. The temperature and pulse must be taken every three hours and recorded, for this may be the only way we have of diagnosing sinus involvement.

As to the use of the antistreptococcic serum, my experience has been that any case severe enough to warrant its use needs a mastoid operation worse than an injection of the serum.

In chronic mastoiditis, like chronic appendicitis, no treatment seems to be of value save the radical or the meato-mastoid operation.

PAPER:

THE RADICAL MASTOID OPERATION.

JOHN JOHNSON KYLE, M. D.

The radical mastoid operation is advocated for the relief of a chronic suppuration of the middle ear and its adnexa. However, Donelan reports a case of "chronic otitis" in which this operation was performed.

The first question which naturally arises in the consideration of the radical mastoid operation is, when does the acute condition end and the chronic stage begin? The stage of the disease may be ascertained, according to William Milligan, University of Manchester, (Journal of L. R. & O., October, 1907,) by the "clinical, histological and bacteriological evidence." The course of an acute otitis media purulenta is somewhat variable. However, a chronic condition is said to begin after the acute febrile symptoms have passed away and the perforation in the drum, through which pus, with or without odor, flows from the middle ear cavity, becomes well formed.

Histologically, in the acute form, we find, according to Milligan, an "extensive shedding of the superficial columnar epithelial elements of the mucosa in conjunction with a copious leucocytosis and the frequent presence of red blood cells in the discharge as chronicity becomes established, the type of the epithelium becomes changed to a much more flattened and squamous variety, whilst leucocytes tend to be replaced by lymphocytes and myelocytes." The greater the number of leucocytes and degenerated columnar epithelial cells and the fewer the number of lymphocytes in the discharge, according to Milligan, the more acute and superficial the inflammatory process and the more amenable to treatment; and conversely, a decreasing number of living leucocytes in the discharge, the discharge still remaining fairly copious and filled

with a number of lymphocytes, the deeper the tissue changes effected, the more chronic the process and the more positive the indication for the radical mastoid operation.

In the acute process, the infection is more often monobacterial, whereas in the chronic process, the infection is usually polybacterial and saprophytes predominate. The chronic cases may, after the division suggested by Heine, be classed as dangerous and not dangerous. We would classify those cases as dangerous, in which there was present evidence of deep involvement of the subcutaneous structures, and in which there were present saprophytes, meningococcus of Weichselbaum, pneumococcus, gonococcus or the Klebs-Loeffler bacillus. Those cases with a chronic ill smelling discharge, with or without cholesteatoma, constant or periodical pain in the region of the temporal bone, labyrinthine suppuration, facial nerve paralysis, meningeal irritation or sinus involvement, are extremely dangerous.

The presence of the streptococcus pyogenes, on acccount of its liability to produce acute exacerbation of the disease with involvement of the sinuses or meninges when present for any length of time, is an indication for the radical mastoid operation.

In those cases in which the infection is monobacterial and free from cholesteatomata, with a large perforation in the drum, especially through the membrana tensa, with or without polypi, and no evidence of involvement of the adnexa of the middle ear, palliative treatment may be all that is necessary.

Briefly, the cases in which the operation is contra-indicated are, according to Laurens, cachectic and advanced tubercular patients, and those cases which cannot be subsequently observed by some one capable, for the dressing must be regularly made until the cure is complete. There are exceptions to all rules, in this as in all things, for instance,—in acute exacerbation of an old chronic mastoiditis with profound febrile disturbance and excruciating pain, the radical mastoid operation is indicated even if the prospect of the ultimate recovery of the patient is impossible. If a few weeks of comfort can be given a patient suffering from a general pulmonary consumption, I think the operation is justifiable.

A great deal of discussion has resulted from the consideration of the indication for ossiculectomy as opposed to the early radical

mastoid operation. Ossiculectomy is unsatisfactory. Ossiculectomy and curettement through the canal are very liable to start up an acute exacerbation of the mastoid disease. If a circumscribed pocket of suppuration exists, ossiculectomy and curettement may cure the disease. Milligan reports thirty-two per cent of his cases in which ossiculectomy was performed, going on to the radical mastoid operation after four weeks to two years. After all, ossiculectomy and curettement is a stab in the dark.

The simple presence of polypi that can be removed with a snare or the application of a fifty per cent solution of trichloracetic acid or chromic acid, in the absence of cholesteatomata, is not always an indication for the radical mastoid operation.

In this, as in other lines of our special work, there are noticed the same progress and ever strife toward the ideal. Since the introduction of the Schwartze-Kuster-Bergmann-Stacke complete radical operation, with or without myringectomy and ossiculectomy, up to the present time, there have been various surgical procedures, methods of treatment and instruments recommended, looking to the simplification and improvement of the operation. I shall briefly describe my own method of procedure without any effort on my part to tell a new story or especially adorn an old one. In consequence of this declaration, my paper will deal with some points in the technique of the operation without any effort to discuss the principal rules of procedure with which all are familiar; in other words, I want to deal with debatable points in the operation as I see them. Many points may impress you as simple and you may find the whole tedious.

Those of you who care for a complete historical retrospect of the operative treatment in middle ear disease, are referred to an address by William Hill, delivered before the British Medical Association, Exeter, July, 1907.

A perfectly antiseptic technique is the first requisite and in addition to the recognized rules of antiseptic precaution, the mouth of the surgeon should be protected with a gauze mask to prevent the spraying of any bacteria from the mouth into the wound, if the operator is demonstrating the different steps of the operation. The use of rubber gloves is advocated by some operators and ignored by others. To the novice, gloves are bunglesome, but to an experienced operator, they are a source of protection and com-

fort. The accumulation of blood upon the bare hands, drying as it does, and the time required to wash the hands free from the blood during and after the operation, are points in favor of gloves, to say nothing of their value in preventing infection from the hands of the operator entering the wound.

The preparation of the head varies according to the sex of the patient. In men it is better to clip the hair from the head and shave the field of operation. In women, clipping the hair will not be tolerated as a rule, and in consequence, shaving the head at the site of the operation is all that can be done.

After the classical curvilinear incision, the periosteum should be elevated with a heavy periosteum elevator, the one designed by Doyen being the most satisfactory to me. It is the consensus of opinion that the surgeon's attention should be directed first to the discovery and exploration of the antrum. Delaissement advocated and practiced opening first the cells of the tip of the mastoid. This practice, however, has long been discarded, and our first attention is directed to the antrum by the shortest and most expeditious route. Many instruments have been devised for this, the chisel, gouge and mallet being the most popular. I wish to call the attention of the Society to Doyen's trephine which I have on exhibition. This instrument was designed by Doyen, of Paris, for trephining the skull but I have used it for trephining the antrum with so much comfort that I have no hesitancy in commending it to my confreres. The instrument consists of a short metal auger with four burrs, one for beginning the trephination, and the other three for completing the operation. After trephination, the chisel and mallet may be used if indicated.

We should bring about as little concussion of the brain in entering into the mastoid antrum, as possible. The concussion resulting from opening into the antrum with the chisel and mallet, where there is great sclerosis of bone, may be more than one might ordinarily believe, and I sometimes think that a certain amount of the nausea and vomiting following the mastoid operation is in a measure due to the concussion from the use of the mallet.

Not infrequently, total ablation of the cells and bone can be done with Rongeur's forceps. With the Rongeur's forceps most of the sharp edges of bone can be curetted away. And this

brings me to the consideration of the different methods of operators in the effort to secure a perfectly smooth bone cavity. A great many operators, in my opinion, waste much valuable time in securing a perfectly beveled edge and smooth walls after the cells have been ablated. I believe it time wasted, and unnecessary manipulation to do other than smooth down the sharp and protruding points of bone, and to secure a perfectly beveled edge to the bony cavity is rather to hamper the formation of a granulating surface than to encourage the formation of granulations. The same surgical care is indicated in ablation of the cells of the temporal bone, as in bone surgery of any other part of the body, and I do not recall any conditions requiring the minute scraping and polishing of bone that I have observed in many operators in performing the radical mastoid operation. A very important point is the removal of loose spicula of bone and loose shreds of tissue. After irrigating with a hot normal salt solution, the wound is usually clear enough for careful observation. In removing the posterior bony wall of the auditory canal, the narrow Rongeur is frequently the only instrument necessary. If the Heath modification is indicated, the Stacke guide should, if employed at all, be used with caution. For the removal of the bony ridge, I have designed the blunt forked chisel. This chisel is very useful in shoving down the posterior bony wall and approaching the facial canal. It is very necessary that we approach the canal closely when we prepare to do the Jansen flap or modification of the Stacke operation, and in my opinion, this is the operation of choice.

Curettement of the inner tympanic wall after removal of the ossicles should be sparingly indulged in. Granulation tissue about the round or oval windows and the meatus of the bony Eustachian tube should be removed with small biting forceps. In many places, the tissue of the cartilaginous portion of the auditory canal is very much hypertrophied and in consequence the flap is thick and requires a large space for securing close appositon to the floor of the excavated mastoid process. Partial closure of the post-auricular wound and packing with gauze are recommended by many, but as a general proposition I believe the post-auricular wound should be entirely closed with a continuous horse-hair suture. If silk sutures are to be used and the case is to be dressed by a physician not familiar with this form of surgery, it

is better to have the stitch holes about three mm. from the line of tissue incision, for by so doing, no skill is required in cutting the stitches with scissors and these should be removed not later than the third or fourth day, stitch scars being prevented by their early removal.

If the cells extend far back and the sinus wall is found to be diseased, it may be necessary to place a small gauze drain in the lower angle of the wound. This can frequently be discarded after a few days. If the dura or sinuses are exposed and are in a healthy condition, I can see no reason why the exposed dura or wall of the sinus should be covered with gauze, as recommended by many, nor do I deem it necessary to pack the middle ear cavity after the labyrinth has been exposed.

After the closure of the post-auricular wound, the question of the treatment of the wound through the auditory canal is next to be considered, and I have about concluded after many satisfactory trials, that the wound should be allowed to fill with a partial blood clot and the auditory meatus closed with gauze. I do not, as a rule, irrigate, nor do I meddle with the wound. The auricle, meatus, and post-auricular wound are cleansed once daily with gauze moistened with alcohol and the auditory canal carefully mopped with a cotton-tipped probe moistened with alcohol, after which the bandage is applied. This simple and non-irritating treatment is continued until complete dermatization of the wound is accomplished and without any effort at grafting. If the case is one which at the time of the operation was filled with cholesteatomatous degeneration, irrigation may be necessary, followed by the application of a saturated solution of boracic acid in alcohol, either in drops or upon a cotton-tipped probe.

Irrigation of the wound with aqueous solutions has a tendency to bring about a water-logged condition of the granulations, which, as far as I know, inhibits rapid repair. If pus is present, irrigation with normal salt solution may be indicated, and if the exudate is that naturally thrown off from healthy granulations, it should be left alone, for this is nature's method of repair.

In children, if adenoids and hypertrophied tonsils are present, which may prolong the post operative suppuration, they should be removed previous to, or at the same time of, the operation. The

bad effect of any diseased teeth or gums should be controlled by the frequent use of antiseptic mouth washes.

For the benefit of those who may have overlooked Hill's address on the "Operative treatment in middle ear disease,—an historic retrospect," presented to the British Medical Association, July, 1907, I have appended the different historical steps in the operation, as he has compiled them.

CHRONOLOGY OF MEATAL OSSICULECTOMY.

1873-1884. Schwartze introduced ossiculectomy, + partial myringectomy.

1885, Kessel performed ossiculectomy + complete myringectomy + removal of annulus tympanicus.

1885, Loewenberg and Stacke (published in 1888) adopted identically the same operation as Kessel.

1886, Sexton in America, and is 1889, at Leeds, Sexton advocated this method at the meeting of the British Medical Association, and showed his instruments.

1889, Stacke introduced postaural inter-meatal atticotomy + Kessel's operation.

1890, Hartmann performed permeatal atticotomy with special chisel + ossiculectomy + Kessel's operation.

SCHEME OF STACKE'S TENTATIVE OPERATIONS, NOW OBSOLETE.

(1) Stacke (I), post-aural intermeatal ossiculectomy + atticotomy (copied incisions of Schwartze operation for extracting foreign bodies and sequestra).

(2) Stacke (II) or Stacke-Woolf, = post-aural intermeatal ossiculectomy + atticotomy by removing inner end only of posterior meatal wall.

(3) Stacke (III) or Stacke-Küster, = post-aural intermeatal ossiculectomy + atticotomy + removal of all posterior wall. This was an inverted "radical;" now almost obsolete.

POST-AURAL OPERATIONS.

1861, Wilde's incision.

1868, Schwartze (I), mastoidectomy. Schwartze (II), = mastoidectomy + antrotomy.

1879, Kûster (I), complete postero-meatal antrotomy.

1879-89, Schwartze-Kûster, or Kûster (II), mastoidectomy + antrotomy + removal of posterior wall of meatus.

1890. (III) Kûster-Bergmann, included all the Kûster (II) + removal of some of superior meatal wall, including exposure of squamous cells + in some instances ossiculectomy and myringectomy.

1891, Schwartze-Kûster-Bergmann-Stacke (otherwise called "the radical Stacke" (III), or, more briefly, "the radical mastoid operation") = mastoidectomy + antrotomy + removal of posterior meatal wall + removal of some of superior meatal wall, including exposure of squamous cells + atticotomy (with or without myringectomy and ossiculectomy).

CONTRIBUTIONS TO THE IMPROVED POST-AURAL TECHNIQUE.

(1) Preserving membranous meatus .....Stacke
(2) Incision along post-auricular groove.....Koster & Stacke
(3) Suture of posterior wound .....Stacke
(4) More radical removal of lower part of "bridge" as far as facial canal .....Jansen
(5) Applying small grafts to bony excavation.....Hessell
(6) Large grafting operation .....Ballance

PAPER:

THE OPERATION FOR THROMBUS OF THE SIGMOID SINUS AND INTERNAL JUGULAR VEIN, OF OTITIC ORIGIN.

FRANK ALLPORT, M. D.

One of the most important sequelae of middle-ear infections is phlebitis or thrombosis of some of the venous drains of the head, and particularly when such pathologic conditions occur, as they usually do, in the sigmoid bend of the lateral sinus. Disease of this nature is found not only in the sigmoid sinus, but also in the lateral sinus proper, the superior and inferior petrosal sinuses, the cavernous sinus, the jugular bulb, the internal jugular vein and its connections, etc. The sigmoid sinus is, however, its favorite location on account of its vascular connections with the tympanic cavity and adjoining cells and its close contiguity to these frequently infected structures. As the jugular bulb is located directly beneath the tympanic cavity, an unusually high dome to the bulb, or necrosis of the tympanic floor, or congenital dehiscences in the floor, will assist in the conveyance of infection. This will occur through the vessels and lymphatics connecting the tympanic cavity with the bulb, and may produce a primary phlebitis or thrombosis of the bulb, which has not proceeded through the usual channels of the antrum, mastoid cells and lateral sinus. These conditions are usually found in children, develop rapidly, and generally demand immediate operation with excision of the internal jugular vein.

I do not believe that actual intravascular phlebitis and thrombosis of the sinus and vein occur as frequently as might be supposed from a perusal of much of the available literature of the day, because a fairly large experience in mastoid work for many years has not disclosed this to be the case. Nevertheless, the disease occurs with sufficient frequency to demand the most careful

study in order that prompt and skilful action may be taken whenever its existence is actually disclosed.

Risk in Sinus Operations.

The surgery of otitic phlebitis and thrombosis has made great progress of late years and many lives have been saved thereby that must otherwise have been inevitably lost. I fear, however, that over-enthusiasm along these lines may have led some thoroughly competent and trustworthy observers to over-estimate the frequency and importance of the subject and to under-estimate the dangers of operative interference. The opening of the sigmoid sinus, for instance (first seriously proposed by Zaufal in 1880), with its possibilities for infection, especially when done through an already infected mastoid cavity (which is, of course, usually the case), is an operative procedure of importance and danger. Yet some surgeons practically advocate its performance for diagnostic purposes where any reasonable suspicion points to an intrasinus involvement. In my opinion the sinus should usually be opened in the face of mastoid disease, either acute or chronic, accompanied by characteristic phlebitic symptoms, such as chills, sweats and wide excursions of temperature and pulse. It should also be done in the presence of a virulent infection, especially of the streptococcus type, a significant differential blood test, etc. Nevertheless, I have seen a number of such cases cured by the removal of necrosed bone, granulations* and pus from the sinus neighborhood. I should, therefore, be inclined to wait a few hours after the removal of such debris, before opening the sinus itself, unless the patient's condition and symptoms are reasonably decisive and urgent in character, and unless an inspection of the exposed sinus renders the presence of a thrombus exceedingly probable.

It must have been the experience of most surgeons that phlebitic symptoms frequently and permanently may be relieved by a removal of neighboring necrosis, granulations, pus, etc., which perpetually infect and reinfect the sinus walls. This fact is especially emphasized by Bergman in his writings and can certainly be substantiated by all operators of experience. In this observation lies an earnest warning against too energetic operative procedures,

* The existence of granulations on the soft sinus wall is regarded by many as beneficial, in that it is nature's obstruction to the invasion of micro-organisms, and many surgeons carefully refrain from disturbing them during the course of an operation.

without quite positive indications. It is sometimes the case that septic symptoms following a mastoid operation are due to pneumonia, typhoid, malaria and other coincident conditions which should be duly considered and properly weighed before proceeding to a sinus operation. It should not be forgotten that, while lives may occasionally be sacrificed by proper conservatism, it is probable that more lives would be lost by injudicious and hasty surgery.

The provisional determination to open the sigmoid sinus is usually formed before the mastoid has been operated on, or after an operation has been performed and several days have perhaps elapsed, during which time the condition of the patient has not materially improved. This resolution is, of course, liable to be changed by existing intramastoid conditions, after the mastoid cortex has been removed, at the time of the primary operation. In all cases where sinus symptoms are clearly present, the bony covering should be freely and carefully removed and the soft sinus exposed for inspection, whether the osseous partition is necrosed or not. Should the inspection reveal an apparently healthy sinus, operative procedures may be suspended (unless the patient's condition is too grave), as sharp sinus symptoms are sometimes produced by a beginning absorption or phlebitis. This may originate in an infected mastoid cavity, relief from which is not infrequently noticed after a thorough exposure and cleaning of this space.

After this has been accomplished and the patient carefully watched for some hours or perhaps one, two or three days, and the sinus symptoms remain the same or grow worse, the sinus should be unhesitatingly opened, even if its local appearances are apparently healthy. Much the same procedures should obtain in case necrosis, granulations and pus are found in the sinus region, for such pathologic products continually bathing the soft tissue tend to become absorbed and to produce the characteristic danger signals. Such debris may be carefully removed and the sinus amply inspected. Should it be found of healthy appearance, one may stay operative procedures a little, unless the symptoms are quite urgent. Many patients have recovered and will recover under such conservative management, and this fact points a significant lesson.

The Appearance of the Uncovered Sinus.

Of course, the entire subject of opening the sinus hinges on the probable existence or non existence of a thrombus, and this can not always be easily determined. A discussion of this topic is too prolonged for the present article, and is apart from the specific subject under consideration.* As the physical appearance of the uncovered sinus, however, must be observed while operating on a patient suffering with sinus symptoms, this phase of the subject may with propriety be here considered, and it may be said that even a physical examination of the sinus does not always clear up the pathology. The sinus is usually bluish-white in color and easily compressible. Nevertheless, the neighboring necrotic bone, etc., may stain and darken the outer coats of the vessel, without the existence of any intramural clot, or a perfectly normal color may characterize a sinus, in whose interior a thrombus may be found.

A sinus may be thoroughly compressible and yet contain a parietal clot, where the obstruction is not yet complete, but a solid, resisting sinus is, of course, a reasonably positive sign of an obstructive clot. The emission of pus from the mastoid foramen is a significant symptom, as it may then be assumed that extradural pus is present in the cerebellar fossa, around the sigmoid groove. I am opposed to the insertion of an aspirating needle into the sinus in order to ascertain the fluidity of its contents. If nothing is withdrawn it is safe to assume the presence of a thrombus, but blood may escape in case of a partially organized clot, and if the vessel is healthy the operator may easily regret having punctured and probably infected a normal sinus; if the sinus is to be opened at all, it is much better to make a complete incision rather than a puncture. The stroking and compression test of Whiting can not be commended, for, while it may facilitate the diagnosis, it can easily displace infected thrombotic particles and set them into circulation with disastrous results.

The Problem of Diagnosis.

It will thus be seen that making a diagnosis in cases of otitic sinus thrombosis is beset with some difficulties and perplexities.

A tolerably complete analysis of the symptomatology of sinus thrombosis, etc., can be found in a communication by the writer in the Chicago Medical Recorder, January, 1906, and in other books and periodicals.

Yet a correct conclusion can usually be reached, especially when a consideration of the general subjective and objective symptoms has been aided by an operative exposure of the sinus itself through the mastoid cavity. Fortunately this (the mastoid operation) is a procedure which should be performed primarily and, therefore, nothing is lost and everything gained when a mastoid operation is advised in a suspected otitic sinus thrombosis, for the cells, etc., may be thoroughly opened and cleaned and the sinus inspected and perhaps opened during one operative procedure.

Should an operation, or at least an inspection of the sigmoid sinus be decided on, in a case of otitic origin, it is only necessary to consider the operative steps in connection with a thorough exposure of the mastoid cavity, for this is at the same time the easiest as well as the best avenue of approach. The mastoid operation should first be completed in all its details, for, in case troublesome hemorrhage occurs, it might be extremely difficult to continue further operative procedures on the bone. In addition, the work on the sinus should be prosecuted under the most aseptic conditions possible, and this can be best accomplished after the mastoid interior has been thoroughly cleaned. As has been previously said, the bony covering of the sinus may be in a necrotic, softened and fistulous condition, surrounded by granulations and bathed with pus.

The Removal of Granulations.

The removal of such pathologic products can be accomplished with comparative ease by curette, chisel, bur or rongeur, extreme care being exercised in all such manipulations to avoid injuring the soft dural covering of the vessel. Some authors advocate the non-removal of granulations, believing them to be Nature's obstruction to the invasion of the sinus interior by micro-organisms. However this may be in theory, I believe that such tissue is best thoroughly removed, not only to place the parts in as clean a condition as possible, but also to facilitate a careful examination of the sinus, which can not be satisfactorily accomplished through a granular mass. In most instances I accomplish this work by means of a strong, narrow rongeur, one blade of which is inserted inside and the other outside of the bony sulcus, being careful to avoid pressure on the underlying vessel and only removing small fragments of bone at a time. Objections have been made that the

slight pressure on the sinus may induce a fracture of its possibly thrombotic contents, which will be swept into the circulation and induce migratory infection. This objection seems like a refinement of technic and a magnifying of possibilities, for, while such an accident might possibly occur, it is altogether unlikely. It certainly would seem to be a better instrument than the chisel, with its possibilities of bone fracturing and splintering, with perhaps a sinus puncture and an embarrassing hemorrhage.

All the necrosed bone should be removed, no matter how far it extends, either in the direction of the torcular or the bulb, and it is well to remove even some healthy bone in order to insure complete elimination of all necrosis (Fig. 1). The sinus should then be minutely inspected, and if its opening is determined on the operation should be continued after the parts have been thoroughly cleaned and irrigated with antiseptic solution. If it is deemed best to await developments the wound should be cleaned, irrigated and dressed as after other mastoid operations.

In case a primary opening of the sinus is determined on either at the original mastoid operation or at a later date, it should be remembered that an ample exposure is absolutely essential to correct surgical manipulations. The sinus must be exposed, therefore, for at least a short distance above its knee and as far downward in the direction of the jugular bulb as possible. This free exposure may be accomplished by a removal of the necrosed bone alone, where such necrosis is extensive in character, or it may be necessary to remove considerable healthy bone in order to render the sinus easily accessible.

Location of the Sinus.

Unless the situation of the sinus is indicated by a fistula, its position is uncertain. In children it is located more superficially and further forward than in adults, and even in the latter its location is by no means fixed. It is usually, however, to be found at a point about one-half an inch behind the posterior bony meatal wall, just back of the antrum, and a thorough mastoid operation will usually disclose its convexed outlines. The sinus should first be exposed by chisel, bur or curette, after which the bone is best removed with a strong, delicate and narrow rongeur, as previously described. The bone should be carefully removed in

small pieces and the operative field examined after each bite of the forceps, care being taken not to wound the sinus, either by perforating its covering by a sharp fragment of bone or by rupturing an already softened dura by carelessly pulling away adherent osseous chips. The latter accident may be averted by, from time to time, passing a small spatula between the soft sinus and its bony covering and gently separating the two.

After the sinus has been fully uncovered all debris, etc., should be thoroughly removed and the operative field abundantly flushed with antiseptic. The hands and instruments of the operator should be recleaned and every effort made to prevent sinus infection. In opening the sinus there is danger of a fatal aspiration of air occurring, to prevent which the patient's head should be dropped low and the operative area flooded with sterile water. The sinus should now be opened by a free incision along its length, being careful not to pass through both walls of the sinus into the cranial cavity, an accident which is by no means impossible. If there is a flow of blood, two tight rolls of gauze or cotton held in fixation forceps (which should be previously prepared) should be placed one above and the other below the incision, remembering that brain substance and not bone lies beneath the sinus, and that the amount of pressure should, therefore, be limited (Fig. 2). Such pads may be held by the operator, or by his assistant, or both operator and assistant may each hold one; they should not be applied, however, until after the sinus has been opened and an opportunity has been given for a burst of hemorrhage to occur and possible thrombotic particles to become expelled. These pads may be lifted alternately and accurate information obtained as to whether the hemorrhage occurs from above or below.

If free hemorrhage occurs from both directions it is proper to assume the absence of a clot, or at least that any existing loose clots have been expelled by the force of the escaping blood. If the hemorrhage is slight or absent from either direction, a clot in that direction is to be assumed. If the vessel is assumed to be healthy, some iodoform gauze, rolled into a cylinder long enough to cover completely the sinus incision, should be firmly placed over the vessel and supplemented by layer after layer of gauze carefully held in place. The other portions of the mastoid cavity should then be packed with gauze and the entire mass of packing held in position by a firmly applied bandage which may usually

remain in position for three, four or five days, when it should be carefully soaked off (to prevent hemorrhage) and more packing and a bandage applied.

The danger of hemorrhage is usually past by the time the second or third dressing is done. In case there is hemorrhage from above and not from below, the sinus should be further incised in the direction of the torcular and more of the bony sulcus removed if necessary, in order that adequate room for further operative procedures should be afforded. In considering thrombi which extend from the sigmoid sinus back toward the torcular, it should not be forgotten that clots extending in that direction are not usually so infective in character as those spreading in the direction of the bulb. The venous current, of course, naturally flows from the head downward, and, while infectious particles may easily pass down with the blood current, they do not so easily pass upward and backward against the current. It should not be forgotten, however, that blood currents in valveless veins seek the direction of least resistance and may easily become reversed on contact with intravenous obstructions. It is, therefore, quite possible that, while a thrombus in the immediate vicinity of the antrum may be highly infectious in character, yet as it extends backward toward the torcular it may gradually become perfectly innocent, and composed of benign accretions of normal blood clots, that form a natural and healthy obstruction to the passage of infectious micro-organisms.

Treatment of the Thrombus.

It is, therefore, questionable just how much such a thrombus should be disturbed, and some authors strongly advocate leaving it alone. I believe that if the clot is hard and apparently extensive, it should be gently curetted at its visible portion, in order to remove its infectious layers, and then sprinkled and packed with iodoform powder and gauze. In case, however, the clot is small, or soft and unhealthy in its appearance, it should be completely removed by the curette until free hemorrhage occurs (it being remembered that quite free hemorrhage may occur, even with a considerable parietal thrombus, or from, for instance, the superior petrosal sinus, though the sigmoid sinus is more or less blocked with thrombotic accretions) even if it becomes necessary to remove more and more of the bony sulcus with a corresponding extension

of the sinus incision. A small-sized curette should be employed for the removal of thrombi, small enough to allow of intrasinus manipulations, and its spoon may, of course, be carried considerably beyond the limits of the bony opening (Fig. 3).

It should not be forgotten that a sinus infection of several days' duration may soften the sinus walls to such an extent as to render their perforation by a curette exceedingly easy; curetting should, therefore, be carefully performed, lest brain infection be thus produced. Ease of manipulation may be much facilitated in all sinus curettements by not only incising the sinus, but by excising a large piece of its outer dural wall, and diagnosis and surgical precision will be greatly aided by exposing the intrasinus openings of the mastoid emissary vein and the superior petrosal sinus. The former will be found in the posterior wall of the sinus at a point about on a level with the floor of the ordinary radical mastoid operation when finished. The latter enters the sigmoid sinus from the cavernous sinus at a point about on a level with the roof and posterior wall of the ordinary radical mastoid operation, when completed; in other words, at about the junction of the lateral with the sigmoid sinus, and passes into the sigmoid sinus.

In endeavoring to get as close to the jugular bulb as possible the surgeon should not press too far forward, as he is liable to injure the facial nerve, as it passes down through the posterior meatal wall on its way to the stylomastoid foramen. Bleeding may easily be stopped by carefully applied gauze rolls. In case free hemorrhage does not proceed from the bulbar direction of the sinus, a very serious condition confronts the operator, for, the blood current of the inferior petrosal sinus entering the anterior portion of the jugular bulb, on its way from the cavernous sinus, constantly playing on the septic end of the thrombus, naturally carries the infectious micro-organisms down into the bulb, internal jugular vein, etc., toward the heart, lungs, liver, etc., thus readily producing sepsis, pyemia, metastatic abscess, etc., which usually result fatally.

It is, therefore, necessary that this end of a thrombus, or a thrombus located in this portion of the sinus, should be as thoroughly removed as possible, in order that the further or general spread of infection may be stopped. This may probably be best accomplished by the curette, which should be gently manipulated until

free hemorrhage from below occurs, which may be controlled by the rolls of gauze, bandage, etc., as previously described. It must not be forgotten, however, that it is usually quite impossible to successfully curette the jugular bulb by simply proceeding downward by way of the sinus. The curette can reach practically as far as the bulb and is then stopped by an intervening wall, an abrupt curve, etc., so that if a complete thrombus extends downward as far as the bulb or further, free hemorrhage will not usually be produced by sinus curettement.

Under these circumstances the operation of Grunert, Voss, Iwanoff and others has been proposed for the thorough exposure of the bulb. Owing to the complications and great danger of producing facial paralysis, hazardous hemorrhage, etc., however, it has never become popular and it is generally thought better to expose, ligate and partially remove the internal jugular vein and then clean out the bulb. It must not be forgotten that this does not always prevent the spread of infection by other channels, and that the sudden stoppage of the venous circulation may force infective particles backward into the meningeal and cerebral veins. While the internal jugular vein is, of course, the main avenue through which infection spreads from the sigmoid sinus to the heart and other organs, nevertheless, even with this principal channel ligated, infection may proceed through the posterior condylar vein, the occipital sinus and the mastoid emissary vein, all of which communicate indirectly with the subclavian and innominate veins, irrespective of the internal jugular. It will be remembered that the posterior condylar vein passes through the posterior condylar foramen and connects the lowest level of the sigmoid sinus with the deep veins at the back of the neck.

Ligation of the Internal Jugular.

Some surgeons invariably operate on the internal jugular before proceeding to open the sinus; others only ligate when free bleeding can not be established from the bulbar end of the sinus; others open and curette the sinus and then wait for a continuance or exaggeration of the symptoms, when the jugular operation is performed. Certainly but few, if any, surgeons would sympathize with the views of Horsley, who only ligates after metastases have occurred, for then it would almost surely be too late. Favorable

reports of jugular ligation in sinus thrombosis need not necessarily be attributed to the ligation, as, for instance, Koerner reports 43 per cent of recoveries without ligation. Still it is probably true that in septic and pyemic cases sinus operations with jugular excision yields better results than when the jugular is not disturbed.

My method of procedure is as follows: The sinus is opened, the head always having been previously lowered, the upper portion is curetted if necessary. If hemorrhage occurs freely from below the operation is finished; if not, the lower end of the sinus is carefully curetted and if free hemorrhage occurs nothing further is done. If free hemorrhage does not occur, pressure pads and a bandage are quickly applied and the jugular is ligated and excised, the bandage then removed, the sinus, bulb, etc., curetted and cleansed and the entire operative field, both in the bone and neck properly dressed and bandaged. The necessity for the operation is greatly emphasized if the cervical tissues just below the mastoid are swollen and painful, and if any hardness and pain can be detected along the course of the vein in the neck, and if general pyemic symptoms are present.

I am convinced that excision of the jugular is not an operation which should be undertaken hastily, as grave circulatory and other disturbances are liable to ensue, and other methods of relief should first be tried, unless the patient's condition is very urgent. The literature furnishes many instances where sudden optic neuritis, edema of the brain, with hemorrhages and softening, thrombosis of other cerebral sinuses, etc., have followed jugular ligation, and it should not be forgotten that a small and insufficient jugular on the other side, totally unable to assume double duty, may be present and precipitate most unfortunate results. As a deduction from these observations Eagleton urges that in cases of sinus thrombosis, with optic neuritis, it should be concluded that the return venous flow is already seriously obstructed and that great caution should be observed in adding to such conditions by ligation.

Excision Rather Than Ligation.

In case it is concluded to operate on the jugular vein it is better to excise most of it rather than merely to apply a ligature, as, if the jugular operation is necessary at all, its septic or pyemic current should be eliminated as much as possible and its poisonous contents removed. Before operating on the jugular the shoulders

should be elevated on a sandbag or something of the kind and the neck and head allowed to drop down, the latter turned away from the field of operation in order to place the neck well on a stretch, so that the anatomy of the parts is well defined and the tissues easier to operate on.

If the patient is a male the beard should be shaved and the skin antiseptically cleaned, care being taken not to disturb and break up a possible clot by rough handling. In the case of a male where it is at all suspected that a jugular operation will have to be performed the beard should be shaved before the mastoid operation is commenced. The neck should now be placed on a stretch and the anterior border of the sternocleido-mastoid muscle clearly located and an incision (Fig. 4) made along its edge extending from its sternal attachment to the mastoid tip.* Bleeding vessels should, of course, be compressed at all stages of the operation so that a clear view of the operative field may always be obtained. The skin and other tissues should be kept stretched apart by blunt retractors. After the skin and superficial fascia have been completely severed the platysma myoides muscle with its oblique fibers can be plainly seen and should be fully exposed and incised along the anterior border of the sternocleidomastoid muscle, which can now be distinctly seen, and which should be fully uncovered by holding back the overlying tissues by retractors (Fig. 5).

The external jugular vein runs down the neck in a line drawn from the angle of the lower maxilla to about the middle of the clavicle and lies between the platysma and the sternocleidomastoid muscle. It crosses the latter muscle at an oblique angle, but does not cross the anterior border of the muscle except at its upper third at the angle of the jaw, where it may be seen, twice ligated, cut between the ligatures and gently retracted from the operative field. If ligation of the internal jugular were the only object to be attained it would not be necessary to touch the external jugular, for the ligature would be applied much lower down, near the cricoid cartilage, but as the intention usually is to excise practically the entire vein, it may be advisable to get the external jugular out of the way. It should be preserved if possible, however, as it performs an important part in caring for the venous blood after the internal jugular is removed.

* Crile and others sometimes cut down on the sternocleidomastoid muscle and expose the vein by separating the muscular fibers.

Retractors should now be placed around the anterior border of the sternocleidomastoid muscle, and the muscle pulled back in order to expose to view the carotid, jugular, etc. This object may be facilitated by tearing and pulling the tissues apart by blunt instruments or the fingers, as care should be exercised that vessels and nerves are, as far as possible, uninjured, a thing difficult to prevent if knives and scissors are employed. The exposure should be made throughout the entire length of the sternocleido muscle from its mastoid to its sternal attachments, for it will be the object of the operation to remove practically all of the vein, and it should, therefore, be completely exposed (Fig. 6).

The deep fascia of the neck will be seen on retracting the sternocleido muscle, and on opening this and throwing it back the common sheath of the jugular, carotid and pneumogastric nerve may be seen. The utmost care should, of course, be taken to avoid wounding these structures, and as far as possible all sharp instruments should be eschewed in performing the delicate dissection. The internal jugular vein, the common carotid artery and the pneumogastric nerve are all encased in a common sheath. At the superior carotid triangle (the usual center of operative procedures) or at a point about midway between the angle of the jaw and the clavicle, the vein lies to the outer side of the artery, with the nerve between and posterior to both (Fig. 8), resembling, as Deaver says, the ramrod to an old-fashioned double-barreled shotgun.

Anatomic Relations.

It should be remembered that while the relative location of those different anatomic constituents differs slightly in various portions of the neck, the vein always lies to the outside of the group and can be distinguished by its bluish-white color, by the fact that it becomes full and prominent on expiration and almost collapsed on inspiration or by pressing on it in the lower portion of the neck, when it will expand with blood (unless thrombosed) and quickly disclose its identity. The common sheath of the vein, artery and nerve should now be opened and also the septum which separates the vein from the artery and nerve, and the vein carefully dissected out, practically throughout its entire extent. The artery is, of course, easy to identify, both from its color and its pulsations, but the nerve may, unless considerable care is taken, easily be raised and ligated with the vein.

All enlarged and diseased glands should be removed. After the vein has been thoroughly liberated from its surroundings, the lower ligature should be placed as far down in the neck as possible, and the upper one high up, as close to the mastoid process as possible, using either ten-day chromicized catgut, black silk or Pagenstecher linen, preferably the first, as there is practically no danger nowadays in the use of catgut prepared by modern methods. Two ligatures are applied at each extremity by means of an aneurysm needle and the vein cut between each pair of ligatures, being careful to include nothing but the vein in the ligatures.

In order to check the breaking off and floating of small thrombotic particles toward the heart, etc., which may easily occur from the necessary surgical manipulations of the operation, the lowest ligature should be placed around the internal jugular vein as early as possible and as soon as a low exposure can be made, after which the regular steps of the operation may be proceeded with. This lowest ligature can usually be best applied at a point just below the angle of the omohyoid muscle. The tributary veins should be ligated, and they may be enumerated from below upward as follows (Fig. 7):

The middle thyroid in the lower third of the neck (Fig. 7, L); the superior thyroid (Fig. 7, H), a little higher up; the lingual (Fig. 7, F), a little below the lower jaw; and, lastly, the common branch of the temporal and facial (Fig. 7, D) veins. Sometimes these veins are so small as not to require ligating, with the exception of the common branch of the temporal and facial veins, which always requires careful attention. In removing the internal jugular it should always be severed above the posterior belly of the digastric muscle (Fig. 7, A) and above the common trunk of the facial and temporal veins, but the ligation need not extend more than half or three-quarters of an inch higher than this junction, as no more cervical veins enter the jugular above this point.

All of the veins which may require ligation enter the internal jugular (practically) in its anterior portion, which if borne in mind will simplify their finding. After all connections are severed the vein may be removed and the parts thoroughly irrigated and cleaned preparatory to closing the wound, remembering that the operation should be performed with all the speed that is compatible with correctness and safety, as such patients should not be kept under

anesthesia any longer than is necessary. Sometimes inflammatory adhesions will so firmly unite the vein to its surroundings that its release will necessitate the expenditure of too much time, to say nothing of the danger of rupturing the carotid artery. Under these circumstances the vein and its tributaries should be split open, cleaned, and the neck wound dressed without permanent sutures.

It is not advisable to undertake to irrigate the sinus and what is left of the jugular, either from below upward or the reverse, as infection can easily pass into the inferior petrosal sinus. It sometimes happens that even after the jugular has been resected the upper ligature removed, and the sinus thrombus removed by curettement, there is no return flow of blood from the bulb, which indicates a thrombus of the inferior petrosal sinus. This is an infrequent complication, as is also a similar condition of the superior petrosal sinus, and should be met by following up the sinus and curetting it. The sinus should be cleaned of all possible thrombotic material by the curette after the jugular ligation, but forced flushing should not be attempted.

It is a good plan after the jugular has been resected to remove the upper jugular ligature and give the inferior petrosal sinus an opportunity to send its blood down through the opened jugular vein, in hopes of flushing away thrombotic debris that might be present. The vein should then be resutured, or, as suggested by A. Knapp, "on the day following the operation, thrombosis having occurred in the jugular vein below the bulb, this vein should be slit up, so that we have access to the bulb from both directions, thus preventing a purulent disintegration of the thrombi in the contributory veins and sinuses." Knapp recommends this procedure, especially in the severest cases.

Treatment of Wound.

The wound should now be drawn together except at its upper and lower extremities, which should be left open for drainage. The lower opening need only be very small, just large enough to admit of a round rubber or cigarette drain, which should extend inside the wound for perhaps two or three inches, and which should be gradually shortened from day to day, until it is withdrawn altogether (Fig. 9). The opening in the upper portion of the wound should be larger and should be either packed with iodoform gauze

or drained with a round rubber or cigarette drain to be likewise gradually removed until compact healing occurs. Many surgeons do not suture this large neck wound, but draw the parts together with adhesive strips, feeling that the patient should be given every chance for his life by an open wound and free drainage. This, of course, involves an enormous scar but, nevertheless, is justified in bad cases, and possibly always. The jugular operation having been completed, the surgeon should now return to the sinus, on which work had been temporarily suspended.

The compress should be removed from the bulbar end of the sinus and all possible clot removed by means of a curette, after which narrow iodoform gauze should be gently but firmly packed into the interior of the sinus as far as possible. Pressure should, of course, be retained on the torcular end of the sinus and the entire mastoid wound dressed as usual, endeavoring as much as possible to separate the sinus from the mastoid packings. The neck should be abundantly dressed with gauze and cotton, and the mastoid head and neck enveloped in a voluminous and firm bandage, which it is best not to disturb for at least three days. Ordinary dressings at intervals of two or three days should follow until complete healing occurs.

Treatment of Shock.

Should symptoms of shock occur during the steps of the sinus or jugular operation relief may be obtained by injections of physiologic salt solution into a vein, the breast or abdominal tissues or the bowels. If an intravenous injection is made, from sixteen to eighteen ounces of the solution should be used at a temperature of about 106 F. If the injection is made into the tissues or bowels from one to two pints should be used at a temperature of about 112 F. At all sinus and jugular operations appliances for making these injections should always be ready for instant use. In case the patient is in a very grave condition it is a wise precaution to give an intravenous infusion of physiologic salt solution before beginning operative procedures. Such infusions are also valuable in cases of general infection to counteract and combat the potentiality of migrating micro-organisms.

EXPLANATION OF PLATES.

Fig. 1.—Exposing the sinus. A, mastoid antrum; B, sigmoid sinus; C, mastoid emissary vein.

Fig. 2.—Compression of the sinus to arrest hemorrhage, and to ascertain the presence of a thrombus.

Fig. 3.—Curetting the sinus. A, mastoid antrum; B, sigmoid sinus; C, mastoid emissary vein.

Fig. 4.—Initial incision for exposing internal jugular vein.

It will be observed in Figures 4, 5, 7 and 9 that the incision in the neck is continuous with the incision over the mastoid process. This point has been brought out in the pictures because in neck surgery where the internal jugular vein is to be removed, owing to a previous mastoid infection, the incision in the neck should be secondary to and continuous with the incision over the mastoid process.

Fig. 5.—Exposure of platysma myoides muscle and external jugular vein. A, sternocleidomastoid muscle; B, external jugular vein; C, platysma myoides muscle.

Fig. 6.—Showing the course of the sigmoid sinus, jugular bulb and internal jugular vein. A, external auditory meatus; B, sigmoid sinus; C, facial nerve; D, jugular bulb; E, digastric muscle; F, internal jugular vein; G, spinal accessory nerve; H, sternocleidomastoid muscle.

Fig. 7.—Exposure of the internal jugular vein, etc.; the vein is about ready for removal. A, posterior belly of digastric muscle; B, spinal accessory nerve; C, occipital artery; D, facial vein; E, hypoglossal nerve; F, lingual vein; G, sternocleidomastoid muscle; H, superior thyroid vein; I, communicantes hypoglossi nerves; J, common carotid artery; K, phrenic nerve; L, middle thyroid vein; M, stylohyoid muscle; N, descendens hypoglossi nerve; O, anterior belly of omohyoid muscle; P, sternothyroid muscle; Q, sternohyoid muscle.

Fig. 8.—Cross section of the neck (after Deaver). A, pneumogastric nerve; B, internal jugular vein; C, common carotid artery; D, descendens hypoglossi nerve.

Fig. 9.—Closure of the wound showing scheme of drainage.

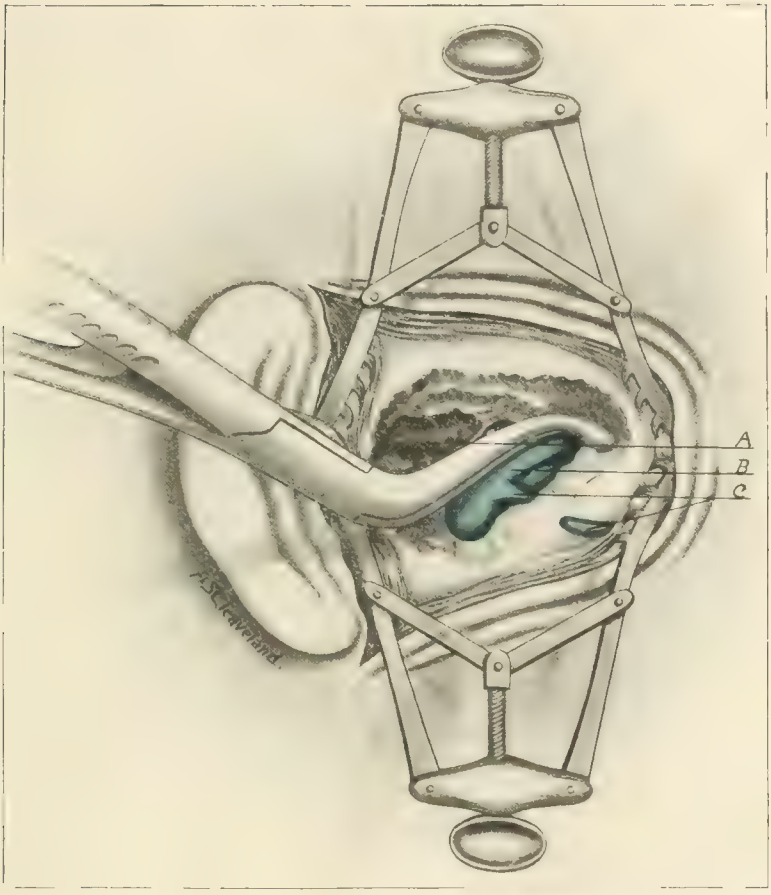


FIG. 1.

Exposing the sinus.

A Mastoid antrum. B Sigmoid sinus.

C Mastoid emissary vein

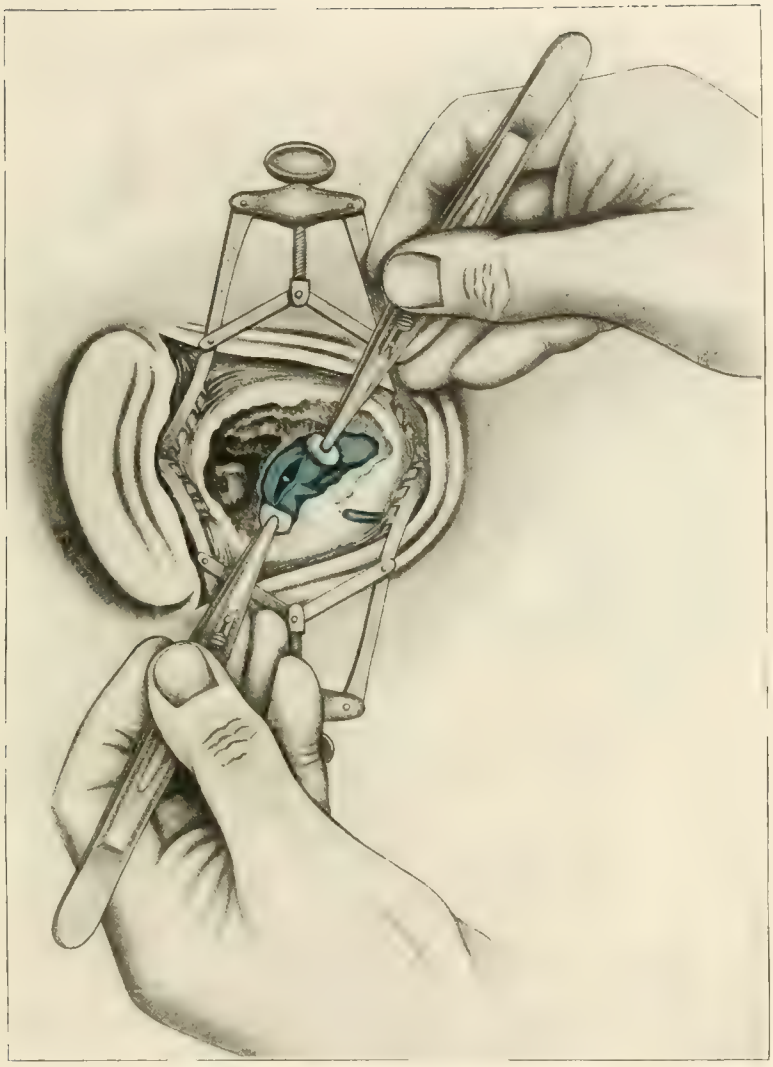


FIG. 2.

Compression of sinus to arrest hemorrhage, and to ascertain the presence of a thombosis.

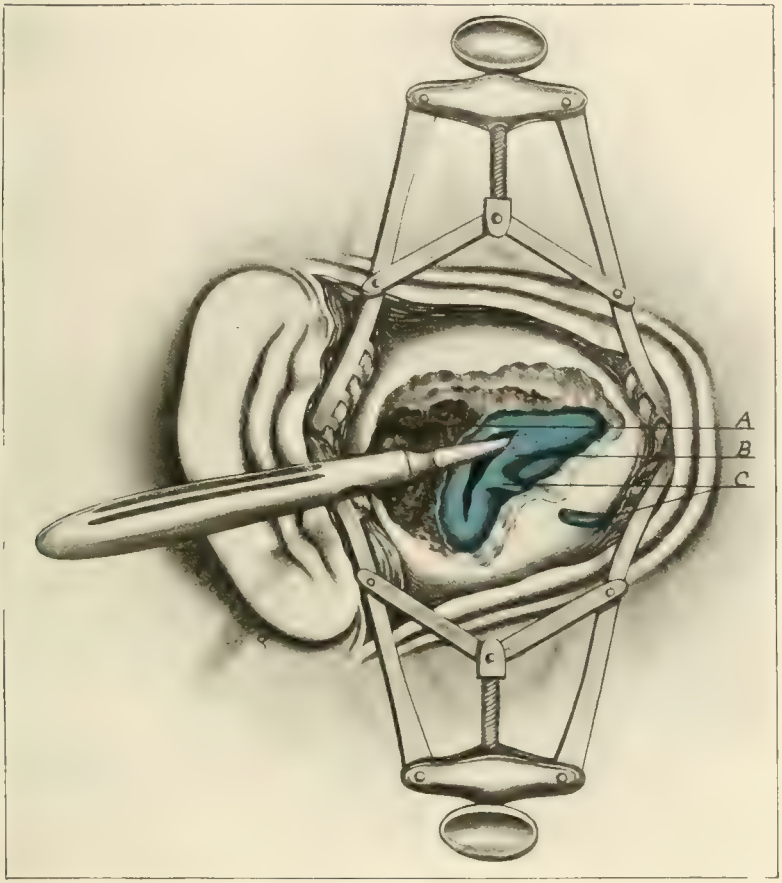


FIG. 3.

Curetting the sinus.

A—Mastoid antrum. B—Sigmoid sinus.
C—Mastoid emissary vein.

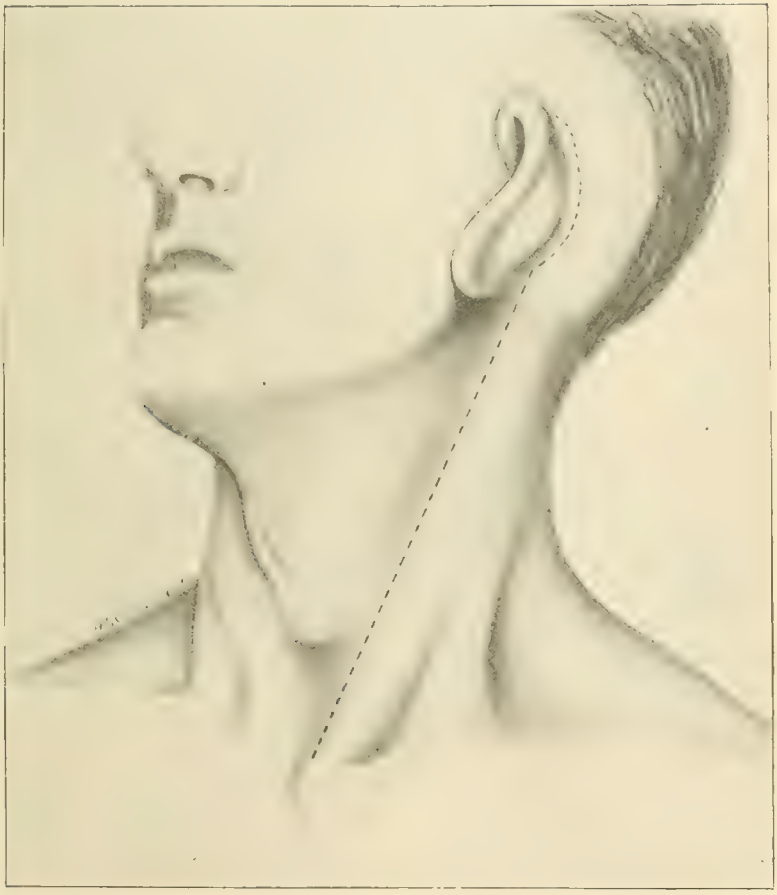


Fig. 4.—Initial incision for exposing internal jugular vein.

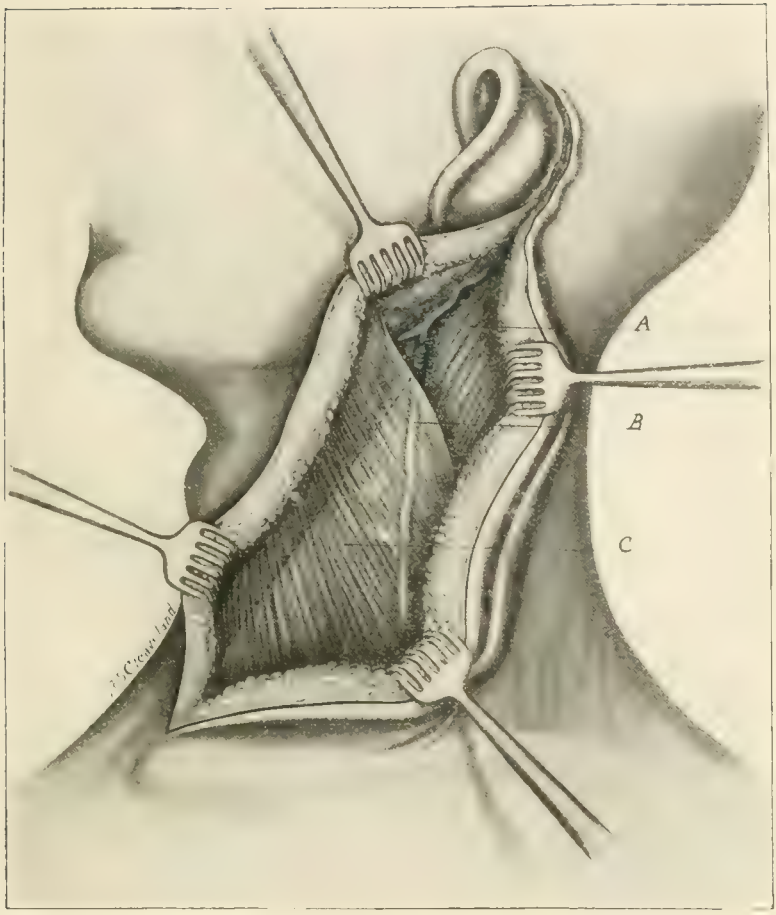


FIG. 5.

Exposure of the platysma myoides muscle and external jugular vein
A—Sternomastoid muscle. B—External jugular vein.
C—Platysma myoides muscle

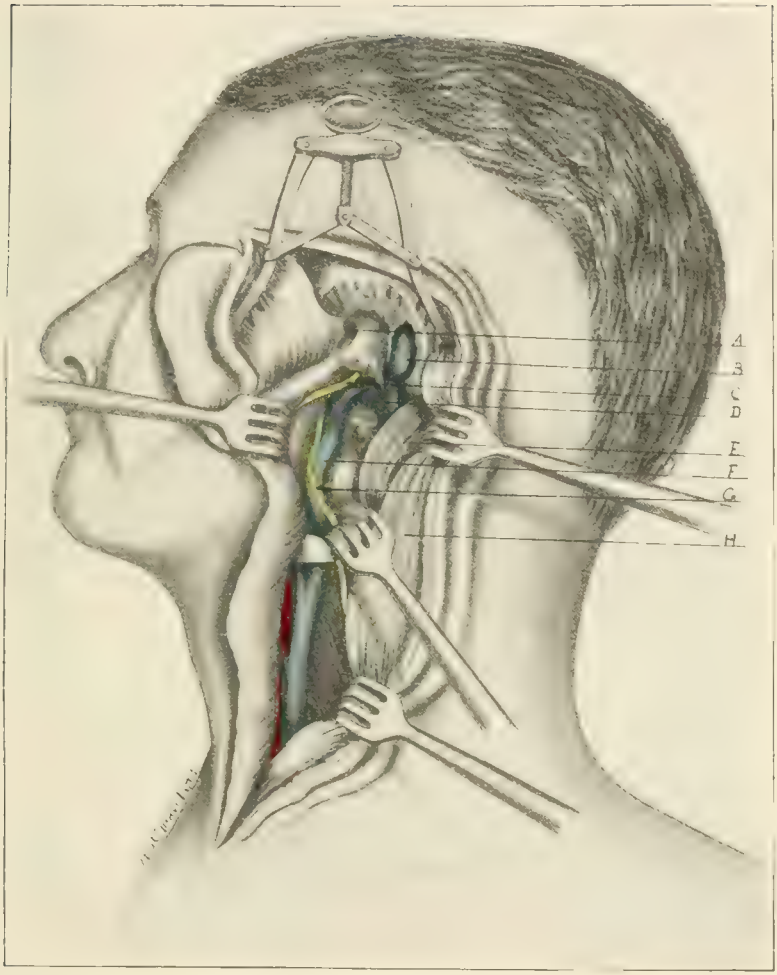


FIG. 6.

Demonstrating the course of the sigmoid sinus, jugular bulb and internal jugular vein.

A—External auditory meatus.
B—Sigmoid sinus.
C—Facial nerve.
D—Jugular bulb.
E—Digastric muscle.
F—Internal jugular vein.
G—Spinal accessory nerve.
H—Sternocleidomastoid muscle.

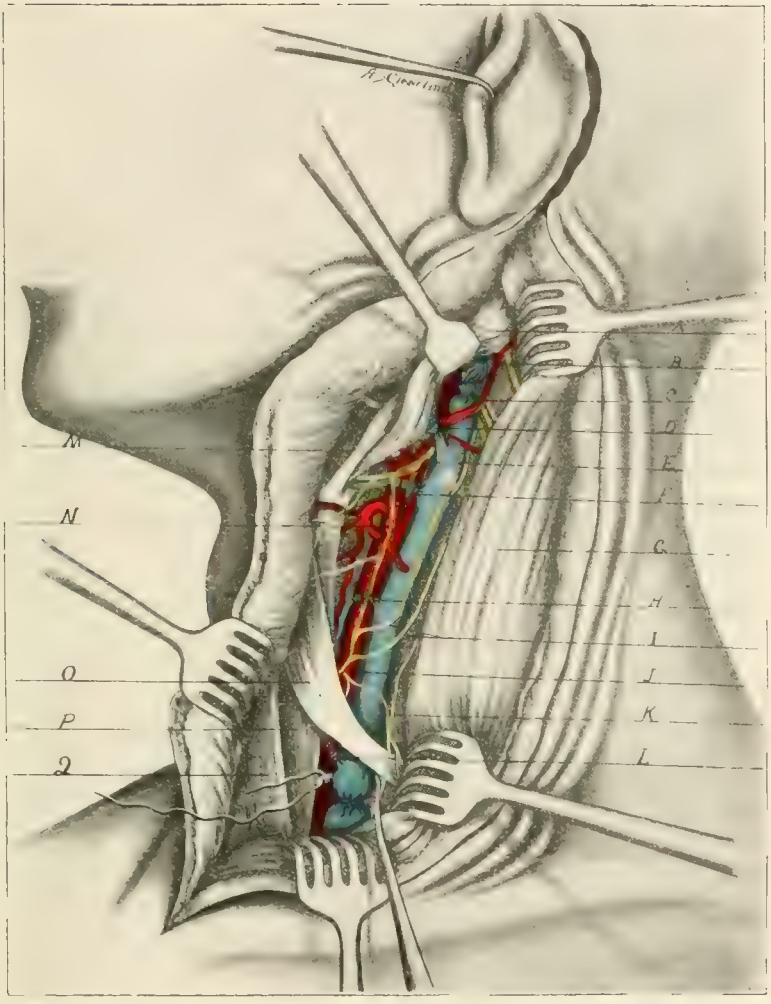


FIG. 7.

Exposure of the internal jugular vein, etc. The vein is about ready to be removed.

A—Posterior belly of digastric muscle.
B—Spinal accessory nerve.
C—Occipital artery.
D—Facial vein.
E—Hypoglossal nerve.
F—Lingual vein.
G—Sternocleidomastoid muscle.
H—Superior thyroid vein.
I—Communicantes hypoglossi nerves.
J—Common carotoid artery.
K—Phrenic nerve.
L—Middle thyroid vein.
M—Stylohyoid muscle.
N—Descendens hypoglossi nerve.
O—Anterior belly of omohyoid bone.
P—Sternothyroid muscle.
Q—Sternohyoid muscle.

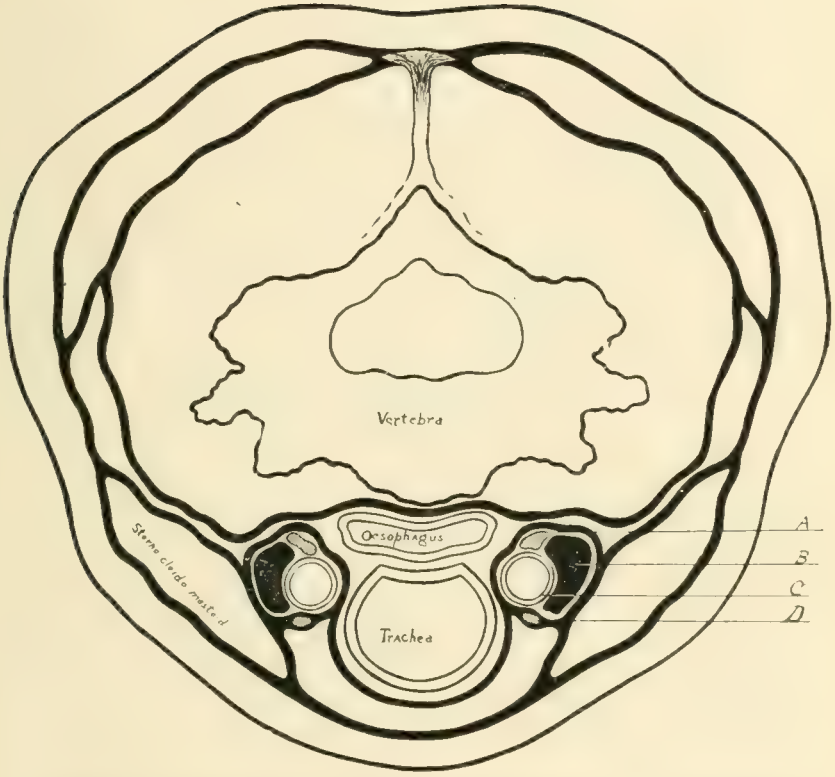


Fig. 8.—Cross section of the neck, after Deaver. A, pneumogastric nerve; B, internal jugular vein; C, common carotid; D, descendens hypoglossi nerve.

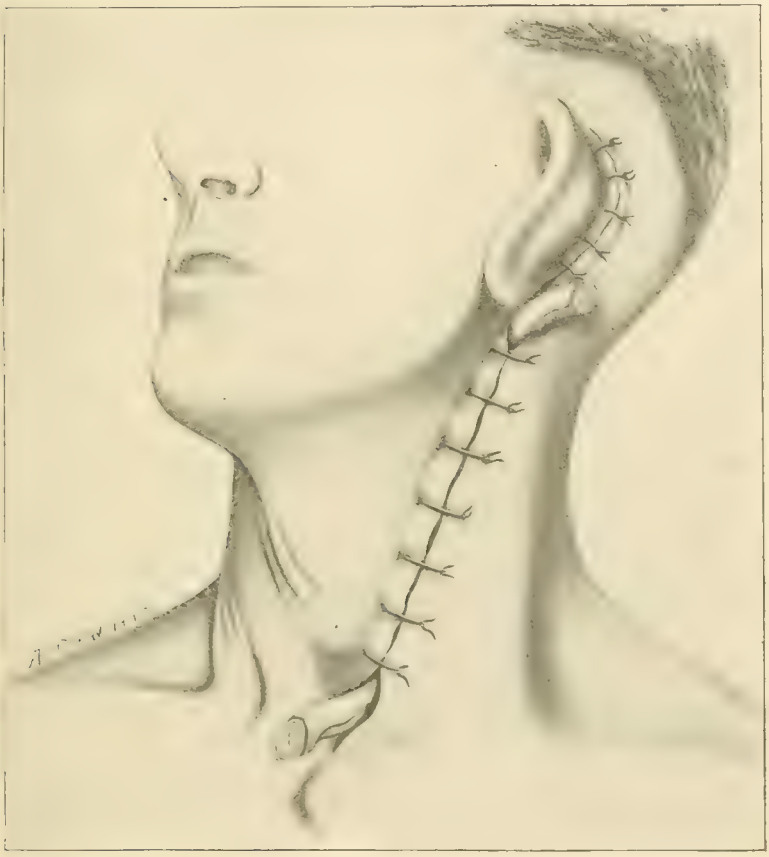


Fig. 9.—Closure of the wound, drainage, etc.

PAPER:

A REPORT OF ATYPICAL AND TYPICAL CASES OF SINUS THROMBOSIS COMPLICATING MASTOIDITIS.

W. R. DABNEY, M. D.

The more cases of sinus thrombosis that I see as a complicating factor in mastoiditis, the more I become convinced that the symptom complex, as laid down in the books,—much as chills, hyperpyrexia sweating, and a rapid easily compressible pulse,—is not always diagnostic of a septic thrombo-phlebitis.

There are a number of acute infectious conditions that are quite frequently manifested after mastoid operations which so closely resemble sinus thrombosis in their early symptoms, that the diagnosis should be made by exclusion, rather than by the symptoms of sinus infection that may be presented.

Pneumonia is not infrequently seen, as a complication after operations upon the temporal bone, and that this disease can very closely resemble a sinus infection—in its symptomatology—is well illustrated in the following case.

Case 1. Child of twelve years. An old chronic otitis media lighted up by an attack of tonsillitis; operation on tenth day after mastoiditis began. At time of operation the temp. was 99° F. and pulse 100.

Operation. Subperiosteal abscess over mastoid and temporal fossa; mastoid empyema extending into the superior meatal cells and cells of the anterior wall of the bony canal; peri sinous extra dural abscess over the arch joining the horizontal and vertical portions of the lateral sinus.

The sinus was not explored, as the history of the case was not very clear, and the patient was not taking the anesthetic kindly.

The posterior bony canal wall was taken down, and Panse flaps stitched into the mastoid cavity. The vertical incision over the mastoid was left open.

Six hours after the operation, patient had a chill which was followed by a rise of temperature to 105° F. and pulse 150. There were frequently repeated slight chills, followed by high temperature and rapid pulse for two days when the characteristic sputa of pneumonia appeared and rendered the diagnosis clear. Auscultation and percussion was negative during this time, and we often find this so, in the early stage of a deep seated central pneumonia.

The pneumonia resolved by lysis, in this case, and the patient was discharged at the end of the sixth week after the mastoid operation.

Acute septic nephritis is another complication that should be considered before we determine that we have to deal with an infected clot in one or more of the venous sinuses of the dura mater.

Case 2. Purulent mastoiditis in a young man complicated by extra-dural abscess above the roof of the antrum and tympanum.

Three days after operation, there was a chill, rapid rise of temperature from 99° F. to 104° F., and exhaustive perspiration. The pulse increase was out of ratio to the temperature, as it was never above 85 per minute; there was partial suppression of urine, and the urinary analysis showed albumen, pus, and cells from the pelvis of the kidney.

The kidney complication cleared up under appropriate treatment.

Case 3. Child 14 years of age. Acute mastoiditis; ear had been discharging for six weeks.

At the operation, we found only a small quantity of pus, but thick bloody serum was in abundance, in all of the cells of the mastoid.

On the sixth day following the operation the patient had a chill, which was followed by a rise in temperature and pulse, and there was free perspiration. The next day there was seen a slight croupous exudate on the same side of the throat as the operated mastoid. There was tenderness along the upper third of the internal jugular vein. This tenderness was, no doubt, due to the involvement of the anterior cervical lymph nodes that overlie the internal jugular, although there may have been a phlebitis of the jugular by contiguity of structure.

A culture from the throat showed the influenza bacillus.

Under aconite, quinine, camphor and aspirin, the symptoms of sinus thrombophlebitis disappeared.

We have seen a number of cases of gastrointestinal disturbances following mastoid operations, which upon a superficial observation could easily be mistaken for sinus infection, but we have only to make the test for indican to clear up the diagnosis, and when it is found, we have but to treat the case accordingly, and the symptoms of sinus involvement disappear with surprising rapidity.

Pulmonary, kidney and liver complications as concomitants of an acute osteomyelitis of the temporal bone, are easily accounted for, when we consider the fact that there is, in this condition, a thrombosis of the arteries as well as of the veins, and when small particles of these thrombi become detached they are deposited as infected emboli in the above mentioned organs, and thus form a nidus of infection that gives rise to the symptoms, which indicate that these organs are involved in a septic process.

Case 4. White female, age 14 years. I was called to see this patient on account of an acute exacerbation of a chronic purulent otitis media.

History.—Both ears have been discharging at intervals for ten years. During the apparent quiescent periods, the patient would remove scales of inspissated pus from her ears; has had most of the diseases incident to childhood, and her ear trouble had its inception in an attack of scarlet fever.

Present illness began a week ago during an attack of pharyngitis; ears began to discharge on the second day; the flow from the left ear was very profuse, while that from the right ear was scant and foul smelling. During the evening previous to my first visit she had chilling of the lower extremities that was followed by gentle perspiration.

Present Condition.—Temp. 100.6° F. Pulse 120. Complains of intense paroxysmal pain in the right ear, the pain being somewhat relieved after there is a free gush of pus; tenderness to pressure applied over the antrum, tip, posterior meatal cells, and mastoid emissary vein; skin over mastoid is glazed and reddened; post auricular groove is obliterated; large perforation in the lower posterior part of drum; decided bulging of inner end of posterosuperior membranous canal.

The streptococcus was the predominating organism in a smear that was taken from the pus as it came through the perforation in the drum.

Operation advised and declined as the father of the child was out of the city and the consultant did not agree with me as to the advisability of an early operation.

December 14th, saw patient at 1 and 9 p. m., and the objective symptoms were more pronounced, although she said that she felt better. When operation is proposed it usually has this effect.

December 15th, word was received from the father that we operate at once if it was deemed advisable. As the child had had several slight chills during the night and was prevented from sleeping by severe pain in the ear and mastoid, it was thought best that we operate as soon as she could be prepared.

6 p. m. Operation. Chloroform. The mastoid was densely sclerosed, and not a single cell was encountered until the antrum was reached, when pus and granulations, as well as cholesteatoma, were encountered; antrum was larger than usual, and the walls were undergoing ulcerative degeneration; the posterior canal wall was chiseled away, and the malleus and incus—which to the unaided eye appeared to be normal—were found imbedded in a mass of fibrous connective tissue that had formed between the drum and the inner tympanic wall; this condition in connection with the granulation formation in the antrotympanic duct prevented free drainage from the antrum into the middle ear and accounts for the paroxysmal pain, as the antrum was not emptied until the pressure became great enough to force the pus into the tympanic cavity. This is a diagnostic point of some importance, as in my limited experience I have always found pus in the antrum when there were repeated attacks of paroxysmal pain that were relieved by a free flow of pus through a large perforation in the drum membrane.

The ebernating process had not advanced so much in the lower two-thirds of the mastoid, but the cells of this region were small and filled with cholesteatoma, constituting a cholesteatomatous infiltration of the mastoid, which is a rare condition in my experience, as this is the second case that I have met with in my practice.

There was a small perforation in the inner table overlying the lower portion of the sigmoid sinus, from which pus was bubbling; perforation was enlarged and a dram of pus liberated. The sinus wall was beginning to soften, and as palpation did not afford us any information, the outer wall was incised between two pressure pads and a fair sized parietal thrombus was taken out of the sinus.

This patient had a slow and tedious recovery, due to the lighting up of an old nephritis.

The above case very forcibly illustrates the fact that we may have infected thrombus, in one of the sinuses of the dura mater, and yet there may not be manifested the symptom complex that we usually expect to see in these cases.

Case 5. This case came under my observation in 1904 at the instance of Dr. E. W. Hill.

White male, age 23 years.

History.—Frequent attacks of earache in the left ear, but there has never been a noticeable fluid discharge from the ear, although he has frequently taken greenish crusts of a pungent odor from this ear. Present trouble began three weeks ago, during an attack of tonsillitis, but mastoid swelling did not develop until two weeks later, that is one week previous to his first visit to my office.

Present Condition.—Temp. 98° F. Pulse 96. Pain deep down in the ear; edema limited to the lower two-thirds of the mastoid. Slight pressure over tip and antrum caused intense pain. Ear was freed of inspissated pus and cerumen. Inner end of membranous canal was found to be sagging and boggy. There was a large sequestrum of bone in the fundus of the canal, which could not be delivered through the external meatus. Mastoid does not transilluminate.

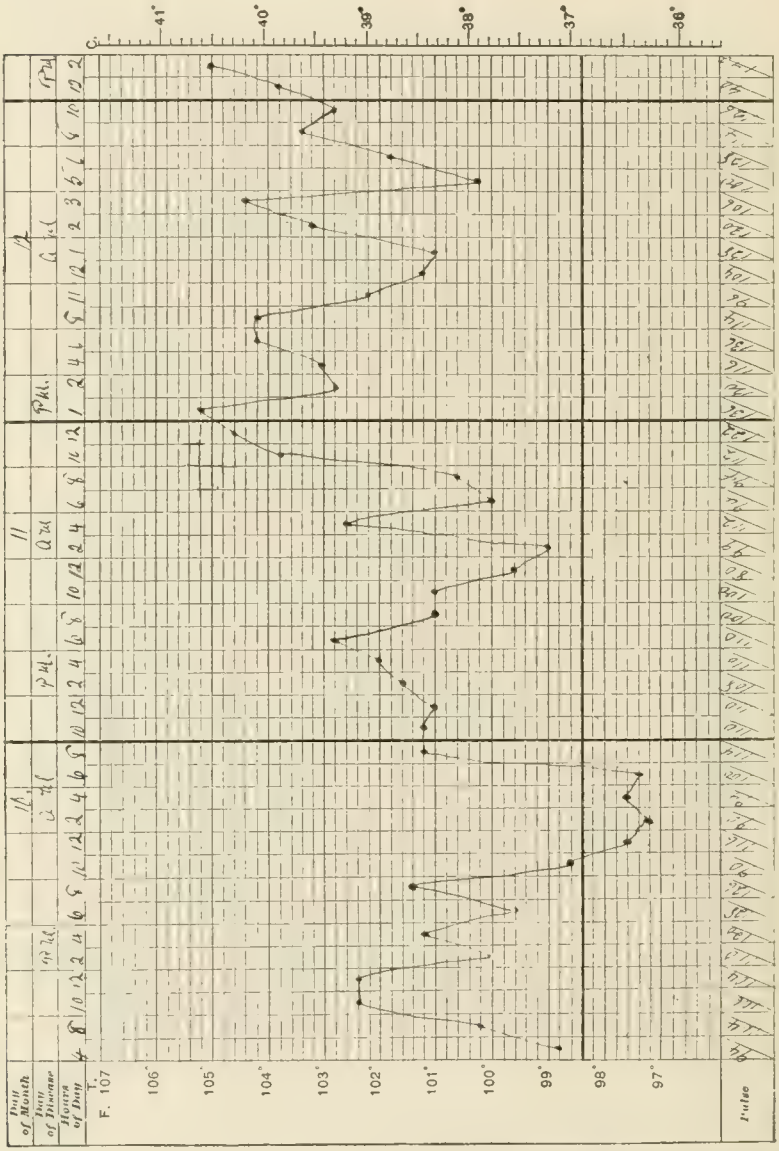
Saw patient at 3 p. m., and his Temp. was 100° F. Pulse 100. He has had a number of attacks of vertigo and several rigors since his morning visit. The hearing power, for both high and low tones, is very much impaired. Advised operation at once, but the young man was under legal age, and the consent of his parents could not be obtained before the following day.

June 10th, Temp. 98.8° F. Pulse 98. Chill during the night, which was followed by free perspiration. The ear is discharging a thin, blood streaked material, from which a smear was taken, and the streptococcus was found to be the predominating organism.

4 p. m. Temp. 100.5° F. Pulse 115. Marked increase in the objective symptoms. The father tells me that the boy had an acute ear condition in early infancy, and that since that time there had been an accumulation of greenish malodorous crusts about the exteral meatus.

June 11th. Operation. Chloroform. Subcortical cells and a large tip cell contained pus; antrum and tympanic cavity were filled

with pus and cholesteatoma. The antral walls were undergoing dry caries as the bone came away under the curette like grains of sand.

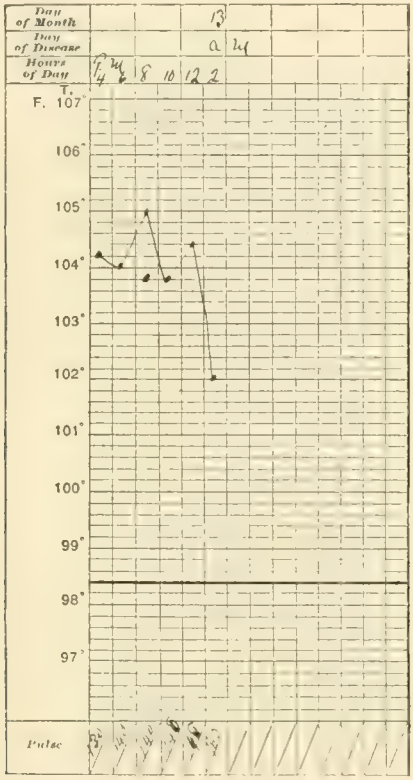


A sequestrum comprising the inner end of the posterior and inferior walls of the external auditory canal was taken out, thus leaving the facial nerve exposed in its vertical portion. The inferior

and anterior walls of the tympanic cavity were absent and the jugular bulb, the internal carotid artery, and the capsular ligament of the glenoid fossa were exposed.

The wound was packed with strips of iodoform gauze and left open.

The young man was relieved of his pain and vertigo by the operation, but the fluctuations of temperature and pulse, and the slight chills were repeated three and four times a day; each chill was



followed by a rise of temperature and pulse and mild perspiration. This condition continued for ten days before the consent of the parents could be obtained for another operation. During this ten days the temperature varied from 97° F. to 101° F. and the pulse from 110 to 140.

As the mastoid wound was in good condition, and the jugular bulb previous to the operation had been in gross communication with the pyogenic material in the tympanic cavity, it was thought

that we had to deal with a primary thrombosis of the jugular bulb, and that small particles of the infected clot were becoming detached into the general circulation and thereby causing the form of bacteremia that was manifested.

On the tenth day following the primary operation the patient was anesthetized and the sigmoid sinus uncovered downward to the bulb. A palpable clot was not discoverable, therefore an aspirating needle of large caliber was inserted into the sinus and a free flow of blood followed but, when pressure was applied to the sinus above the needle, the flow immediately ceased. Pressure was maintained and this portion of the sinus was laid open with a small knife; a curette was introduced, and particles of softened clot were removed but we were unable to re-establish the circulation. It was deemed best to resect the jugular before resorting to further manipulations to free the sinus of the thrombus.

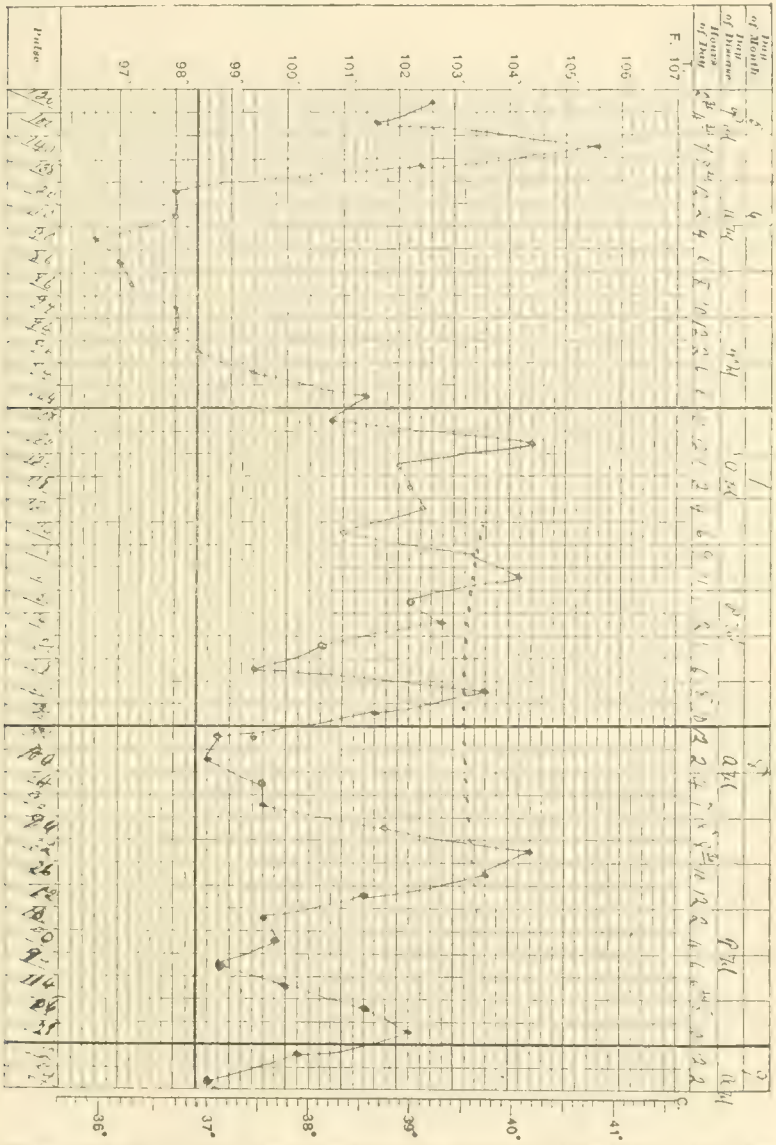
After the vein was excised the sinus was incised down to the bulb and a great deal of disintegrated clot was taken out. A gauze drain was inserted upward into the bulb, in order that it might bring away any softened material that had escaped our efforts.

The temperature and pulse became normal within four days after the sinus operation and remained so throughout his convalesence.

This case serves to illustrate the difference between an actual involvement of the labryinth by the pyogenic process and a labyrinth disturbance due to pressure transmitted through the fenestra in the inner tympanic wall, as the labyrinthine symptoms disappeared as soon as the pressure was removed by operation.

The low form of bacteremia that was manifested in this case may be accounted for in several ways: 1. That the clot in the bulb and lower part of the sigmoid sinus was disintegrating slowly, and thus such small infected emboli were detached into the blood stream that the phagocytes were able to perform their function as scavengers before the extreme constitutional disturbances, that are usually seen in a bacteremia, could be manifested. 2. It is quite possible that there may have been an attenuation of the organism responsible for the infection in consequence of the long continued course of the suppuration, just as we see it in chronic gonorrhea. 3. The individual equation of susceptibility and immunity or resistance to infection must be considered in a given case as the same conditions of infection in an individual of good

resisting powers would not produce the violent train of symptoms that would be manifested in an individual who was greatly debilitated before the infection.



The wound in the neck healed by primary intention, and the reason for this, I think, was due to the method of technic that was followed in the resection of the vein. The technic of the pro-

cedure was as follows: A heavy pad of gauze was passed under the vein, preliminary to cutting it between the double ligatures, in order to prevent septic material in the severed ends of the vein from infecting the wound in the neck.

Before the stumps of the severed vein were dropped into the neck, their intima were cauterized with carbolic acid and alcohol, just as the general surgeon does the stump of the severed appendix before he returns it to the abdominal cavity.

I have followed the above technic in several jugular resections, with the most gratifying result as to primary union of the cervical wound, in cases that were manifesting a high degree of bacteremia. Opsonic treatment would be of great value in this class of cases.

Case 6. White male, aged 16 years. I saw this case in consultation with Dr. W. H. Metcalf, at 2:30 p. m., Sept. 5th, 1907.

History.—Discharge from left ear since early childhood as a sequel of scarlet fever. There has been pain back of the ear on numerous occasions, when the discharge from the ear would cease temporarily.

Present trouble began about ten days ago during an attack of tonsillitis. He has had a number of mild chills and his temperature has fluctuated between 100° F. and 102° F. He had a very hard chill this morning, but his temperature was not taken until 1 p. m. when it registered 101° F.

Present Condition.—Temp. 102.6° F. Pulse 130. Resp. 22. He has an anxious drawn facial expression, that is suggestive of fear; skin is of a muddy yellow color; tongue is heavily coated; breath is foul; speech is slow and voice tremulous; there is an annoying dry cough, but auscultation and percussion are negative. Has constant pain over entire left side of the head but it is most severe in the mastoid region; he is extremely sensitive to light pressure applied over the tip, antrum, emissary vein and along the course of the internal jugular vein. There is edema, beginning about two inches above the external auditory meatus and extending downward to the clavicle. This edema is particularly noticeable in the occipital triangle, and there is exquisite tenderness to pressure applied in this region.

External canal is full of thick foul pus; drum membrane, malleus and incus are missing, and the inner tympanic wall is covered with polypoid granulations. The streptococcus was found to be the predominating organism.

There is a slight Babinski reflex and Kernig's sign is fairly well marked as the angle of extension is about 120°; vision is indistinct and ophthalmoscopic examination revealed a double optic neuritis. The patient readily recognizes articles handed him and understands the purpose for which they should be used, but he displays considerable difficulty in naming the articles submitted to him.

There is a stable mydriasis of the left pupil and the right one responds very sluggishly.

Diagnosis.—Mastoid empyema, sinus thrombosis, and a provisional diagnosis of meningitis with brain pressure due to an extradural or subdural pus collection.

Admitted to hospital at 4 p. m. Temp. 101.6° F. Pulse 116. Chill at 6 p. m., lasting 15 minutes. 7 p. m. Temp. 105.5° F. Pulse 140. Resp. 28.

Operation began at 10 p. m. and the Temp. was 102° F. The usual mastoid incision was later supplemented by a posterior incision extending to the inion. Opening the cortex set free a thick, dark, offensive fluid that was under considerable pressure as numerous bubbles could be seen in the fluid as it gushed out; large antrum was filled with cholesteatoma; inner table overlying the vertical limb of the sinus was completely eroded and the outer wall of the sinus was covered with granulations from which pus was exuding; granulations were removed and a perforation was found in the wall of the sinus; enlarged perforation and a disintegrated clot was mopped out of the sinus; ulceration of visceral wall of the sinus at the knee; ulcerated part of the sinus wall was cut out with small scissors and about three drams of pus was evacuated, which exposed an area of superficial cerebral necrosis at this point. This is about the region of the naming center as pointed out by Mills, and possibly accounts for the hesitation of the patient in naming objects.

On account of the extensive softening of the clot, in the sinus, it was thought best to ligate and excise the internal jugular vein before attempting to free the sinus of the disintegrated thrombus. The jugular was found to be thrombosed to one-half inch below the facial vein, the latter vein also contained a clot. The jugular was excised below the lower limit of the clot and the stump of the vessel was stitched into the upper angle of the cervical incision.

Sinus had to be uncovered to the torcular before the posterior axis of the softened thrombus could be reached. This sinus was next curetted in the direction of the bulb but a return circulation was not established. A stream of normal saline solution was introduced into the sigmoid sinus in the direction of the jugular bulb and particles of softened clot washed out of the end of the jugular that was stitched into the neck wound. The superior petrosal sinus was opened inward as far as the internal auditory meatus without establishing a return flow.

The posterior osseous canal wall was taken down and the middle-ear freed of granulations. Posterior horizontal incision closed and wound packed with gauze. Lower two-thirds of neck wound closed but it suppurated and had to be re-opened.

Sept. 6th, 6 p. m. Temp. 99.4° F. Pulse 84. Chill at 6:30 p. m. Chill at 10:30 p. m., following which the temperature was 104.2° F. and pulse 130.

Sept. 7th. Chill at 8:30 a. m. followed by a rise of temperature to 104° F. and pulse 140. During the night he complained of pain over the upper portion of the left lung; the respirations are shallow and increased in frequency; large moist rales heard on auscultation and there is prune juice expectoration; has developed a diarrhea and the stools are extremely offensive.

Dressing changed and the entire wound found covered with pyogenic membrane; all stitches taken out, and wound swabbed with turpentine; hot compresses of permanganate of potash solution ordered which were to be changed every twenty minutes. Chill at 7:30 p. m. and one hour afterward the temperature was 103.6° F., pulse 160.

Sept. 8th. 7 a. m. Temp. 99.5° F. Pulse 96. Chill of thirty minutes' duration and the temperature was registered 104.5° and pulse 126.

Slight edema and discoloration of the lids of the left eye.

Sept. 11th. For the past forty-eight hours the temperature has fluctuated between 98° F. and 102° F., and the pulse has varied from 96 to 116. Patient has been exceedingly restless and at times wildly delirious; Kernig's and Babinski's signs are positive and there is some retraction of the head; edema and discoloration of the lids of the left eye have increased.

Sept. 12th. Exophthalmos of the left eye; edema of lids of right eye; projectile vomiting several times during the day; unconscious at 9 p. m. and died six hours later.

Autopsy not permitted.

This patient was given 140 cc. of streptolitic serum, and 12 hypodermic injections of Declat's preparation of phenic acid besides the usual remedies that are in vogue in the treatment of pneumonia.

This case as well as the preceding one serves to illustrate the fact that in an acute exacerbation of a chronic purulent otitis media in which there are symptoms of mastoiditis we should resort to radical operative interference at once if we wish to conserve the interests of our patient. They also serve to show us the extensive destruction that may take place in the temporal bone without the manifestation of subjective symptoms, and from the findings at the operations upon these patients I am of the opinion that chronic suppuration of bone had been in progress for months if not years. Every surgeon who has had a wide experience in dealing with chronic suppuration of bone, has often been surprised at the mildness of the symptoms and the advanced pathological conditions that he finds at the time of operation.

On the day following the operation the young man appeared to be better, inasmuch as the pain was relieved, and he was able to name objects readily. The latter condition would indicate that the naming center had not been destroyed, but as has been suggested by Mr. Ballance, the connecting fibres between this center and the speech center were obtunded to a certain extent by pressure from the subdural abscess.

Cases like the one under discussion, which manifest symptoms of meningitis before operation, do not offer a very favorable prognosis, and in this case as the Babinski and the Koernig signs were positive, we were fully warranted in assuming that we had a complicating meningitis.

As this patient had an annoying cough previous to the operation, although auscultation and percussion were negative, I cannot but conclude that a metastatic pneumonia was developing at that time, as clinicians of experience in diagnosing diseases of the lungs tell us that it is often impossible to diagnose a central pneumonia by auscultation and percussion, and that they have to wait for the appearance of the characteristic expectoration.

I am well aware that, during the manipulations that are necessary to clear a thrombosed sinus, one may dislodge a portion of the infected clot into the general circulation, and that it may be carried by the blood stream to the right heart, and thence to the lungs, to form the focus for a metastatic pneumonia, but I cannot concede that this occurred in the case before us, as the clot in the bulbar end of the sinus was not disturbed until after the internal jugular had been excised, and pressure was made on the opposite jugular while we were clearing the torcular end of the sinus.

The foul smelling purulent stools that the boy had would indicate that his diarrhea was due to a septic enteritis and not to an excess of carbon dioxide in the blood which is the cause of the frequent loose stools in the late stage of pneumonia.

The clinical symptoms of exophthalmos, edema and discoloration of the lids of the left eye, and the later manifestation of the same symptom in the right eye in connection with the fact that we were unable to get a return flow from the superior petrosal sinus, would amply justify us in concluding that there was a septic thrombo-phlebitis of both cavernous sinuses; this being the case, it is quite easy for us to see how the infecting element could be carried to the lungs through the right internal jugular.

It is in cases like this that the diagnosis of a complicating brain abscess is rendered extremely difficult, if not impossible, as the symptoms of bacteremia so predominate that they obscure the characteristic signs of low temperature and pulse that are seen in abscess of the brain.

DISCUSSION OF THE SYMPOSIUM ON SUPPURATIVE DISEASES OF THE TEMPORAL BONE.

Dr. O. J. Stein: With reference to the classification of the causes as given by Dr. Sauer, you can readily see that the list of causes is large, and we do not stop, as a rule, to analyze the various causes in the cases that are brought to us. Frequently, many of these cases come to our clinics and we have not got the time to give proper thought to the causation, and on that account we miss a great deal of valuable information as to the real cause or causes in the majority of cases. But from this excellent compilation of causes as given by Dr. Sauer, we can readily see that in the acute cases a germ disease is the fundamental exciting

cause, and aside from that the predisposing causes are great factors.

A point I want to make is that in infants (and I do not think Dr. Sauer referred to it,) or very small children, where we find the presence of an acute suppurative process, or the symptoms of a subacute condition, not brought out very actively, we are at a loss many times to know the real cause, and I have been brought face to face with this proposition of late in trying to find out what was back of some of these acute processes in small children. It occurred to me, as it has doubtless occurred to other observers, that possibly it might be of gonorrheal origin. I have an excellent opportunity to study this phase of the subject, although I have not gone into it thoroughly, from the information received from the attending physician of a maternity hospital in Chicago, who is going into the subject now. We have found that in very many small children, shortly after birth, if we examine into the causes, we can demonstrate the objective symptoms of ear disease. Subjectively there are no manifestations. We may take a series of many cases, and if we examine them we will find pathology in 95 per cent in subjective or objective findings. We have had evidence of ear trouble where there was no subjective disturbance whatever. It occurs to me that infection from these mothers in these maternity hospitals, particularly of that class where we find these gonorrheal subjects, might be the real causative factor, anyhow producing pathologic changes in the middle ear within this area of embryonal tissue, which lays the ear more liable to an active inflammation under some exciting or secondary cause. We have made a few tests and have demonstrated the gonococcus in the secretions. On the supposition that that may be the exciting factor and also a contributing cause, in later cases I have suggested the employment of the silver treatment, just as we use it in the eyes of the new-born, and in all children with acute suppurative processes, or in the presence of the exanthematous diseases, I have always recommended the instillation of the silver in the nose, after the suggestion made some years ago by [illegible]. It is a good method of prophylaxis in these cases, particularly if there is such an exciting factor as the gonorrheal germ which is causative of the suppurative processes in the ears of children.

Dr. Wm. L. Ballenger: I want to call attention to one etiologic factor which the first essayist failed to mention, and which, I think, is of great importance, and that is, ethmoiditis and sphenoiditis. I believe these are very frequent causes of the perpetuation of an otorrhea. I believe that in all these cases in which we attempt to close the Eustachian tube and fail, oftentimes the reason of our failure is due to the fact that we have overlooked the existence of ethmoiditis and sphenoiditis. Ethmoiditis and sphenoiditis will prepetuate epipharyngitis and this extends by continuity of tissue to the Eustachian tube and to the mastoid cells, and perpetuates a chronic suppurative condition which might otherwise be easily cured.

Another point I wish to mention in connection with the first paper is the intranasal operations which cause middle ear suppuration and mastoid suppuration. I have personally observed a number of cases in which the failure to remove completely the partially severed middle turbinate has been followed by acute mastoiditis. I have found that one of the most difficult things for me to do is, after severing the middle turbinate or attempting to do so, at least, to find a large portion of the turbinated body left. I thought I had discovered before how I could always find it but I have in the past, in a few instances, left a portion of the middle turbinate for a number of days, and in one or two instances for two or three weeks, before I could locate the partially severed middle turbinate, and in a few instances I have seen an acute otitis media following. When we do intranasal surgery, it is important that we remove all the tissue that we attempt to remove. We should not leave fragments as we do in performing adenoid operations. We have hemorrhage which we would not have if we removed all the diseased tissue.

As to the question when an acute process becomes chronic, it is one that has puzzled me for a long time. I believe the safest plan is to classify the cases as chronic whenever permanent tissue changes take place. Pathologists tell us that about the end of the fifth day there is a permanent deposit of the round cells, which finally become spindle or connective tissue cells, and from that time on the case begins to assume a chronic form, and will in many instances require treatment suitable for chronic cases. I do not mean to say that I would advise radical operation

of any type in all cases five or six days old; but I do say, at that time the case begins to assume a chronic form.

Personally, I wish to mention a case of erysipelas which I saw following a mastoid operation. It followed perhaps about four weeks afterward, in which the question of diagnosis came up. The man had chills and rigors, and a high temperature, with remissions such as we find in sinus cases. The man had an erysipelas affecting the cheek upon the side operated, and by waiting it was found that the high temperature was due to the erysipelas and not to a sinus involvement, as was suspected at the time. It is an interesting case, in that it simulated the general symptoms of sinusitis.

Dr. J. A. Stucky: There are so many things in the papers that have been read that it is difficult for one to know just where to begin and where to stop.

In regard to the first paper, I was a little surprised at the statement made by Dr. Sauer that 15 per cent of these mastoid cases, without middle ear involvement, were tubercular. I have had quite a number of cases in which there was non-suppurative disease of the middle ear, but a decided mastoid involvement, and none of these proved to be tubercular.

One point I want to emphasize particularly is the causative relation of suppuration of the nasal accessory sinuses in the production of otitis media, especially the ethmoidal suppuration, and I want to emphasize with a good deal of feeling what Dr. Ballenger said about leaving the tags of the middle turbinate. I am not convinced that it is the best plan to remove the whole of the middle turbinate, but a third or one-half of it, without leaving any of the tags, leaving a smooth surface. As to Dr. Wales' paper in reference to the lesser absorptive activity I think it is due to the fact that we have chronic sepsis in these cases more frequently than we suspect. In examining them pretty carefully we find they have a low form of chronic septicemia.

He mentioned otitic dyspepsia. I am glad he mentioned that because it is new to me. But I have had three cases of chronic enteritis and gastroenteritis in children in the hands of the best men in central Kentucky that have been relieved of chronic suppuration of the middle ear, and the relief came after a radical exenteration. I believe that there is a bone regeneration following the mastoid

operation, if we succeed in saving a good deal of the periosteum. Perhaps some of my cases are not old enough for me to demonstrate this, but in some of them that are seven and eight years old I am convinced that there has been bone regeneration. It is too hard to be fibrous.

In regard to packing after the mastoid operation, I have almost ceased to do so after the third day. I remove the tube and dressing after the first twenty-four or thirty-six hours, and then do no packing.

As regards infection, I have had a run of cases lately which nearly gave me nervous prostration. I believe the grip bacillus causes more trouble, or as much trouble, as the streptococcus. I had a case recently of influenzal bacillus infection which made my hair curl. I have never seen but one Friedlander bacillus infection, and I would like to ask the essayist what he thinks of that. McKernon has laid a good deal of stress on that. The pneumococcus and influenzal bacillus have given me more trouble than the streptococcus.

Dr. R. Bishop Canfield: I would like to say in regard to the last statement of Dr. Stucky, that epidemics of influenza bring about destructive conditions. I think it is rare in these cases, however, that the influenza bacillus is isolated. The condition is, as a rule, a mixed infection.

Dr. Stucky: I would like to ask Dr. Canfield if he does not believe the influenza bacillus is the cause of much of the serious trouble we are having?

Dr. Canfield: The danger lies in the mixed infection. Whether the virulence of the influenza bacillus is accentuated in its combination, or whether it is the virulency of the streptococcus that is accentuated I do not know. When we get to epidemics of la grippe and mixed infection of the throat, we have a serious condition to deal with.

In regard to the regeneration of bone, I have had an interesting case recently in which, six weeks after the first mastoid operation, I was compelled to operate again and found the mastoid cavity two-thirds filled with soft bone. The case turned out to be tuberculous.

Dr. Allport: Was it bone?

Dr. Canfield: Yes, sir. It was demonstrated by a pathologist to be bony tissue.

In regard to otitic dyspepsia, I would include among the causes of the summer diarrheas of children not only suppurative otitis, but serous otitis occurring without symptoms. Through the kindness of Dr. Dock, I have had an opportunity to examine children who never had any ear symptoms, but had had intractable cases of diarrhea or disturbance of digestion, who were relieved after a simple paracentesis, and one or two of them after inflation. It seems to me that serous conditions in the ear, those which are not proven to be infected, have been due to intestinal disturbances in children.

I beg to differ with Dr. Wales when he says that in sinus thrombosis the sinus wall may change. I have seen several cases of sinus thrombosis where the sinus was perfectly healthy, so far as I could tell.

As to the point whether granulations are protective or destructive, according to whether they are infected or not, I think that is an important point. If they are infected, or if they cover infected bone, they may be considered destructive. On the other hand, if their base is healthy, they are naturally protective.

In regard to the danger of different infections, I think the point has not been sufficiently brought out that a great deal depends upon the character of the mastoid. For instance, in a sclerotic mastoid, with a fairly thick inner table, a streptococcus infection need not be so dangerous as a much milder infection where the table is so much thinner.

Dr. Joseph Beck: As to the cause of middle ear disease, I do not think the author of the first paper mentioned cholesteatoma as a pathologic entity which by processes of pressure and destruction involve the middle ear. It is not a secondary cholesteatoma associated with chronic suppuration, but purely an external condition.

Another cause is actinomycosis. I have had the good fortune of seeing a case of primary actinomycosis of the middle ear and of the mastoid process, and it is well to bear that in mind. These cases are not so rare, and characteristic points which anyone can see at the time of the operation are greenish granules of pus, which are the actinomyces.

Recently Dr. Bevan, of Chicago, has had a group of cases of glanders in the neighboring city of Milwaukee, in which his assistant, who worked in these cases, found that the glanders involved both the middle ear and mastoid process, as demonstrated microscopically or bacteriologically. Again, Dench and Dabney speak of osteomyelitis and cholesterin formation in connection with involvement of the mastoid process, and those things should be mentioned under the pathology and etiology.

One of the speakers called attention to the point that there is a chronic sepsis in these cases, and that is the reason they do not get well. I am inclined to believe that it is the lack of the resistance substance in our bodies that is known as opsonins. I hope to be able to bring that point out in my paper this afternoon. It is the only term that is used.

In regard to bone regeneration after a mastoid operation, I will say that recently I showed Dr. Allport a mastoid case that I operated on seven times, three times on one side, and four on the other, and I had a good opportunity to tell whether bone reformed or not. If you were to see that girl's mastoid region, you would say it is as smooth as yours. There is no retraction. It is filled with a mass. I have removed these masses, and there is not a bit of bone except that portion adjacent to the periosteum which I excised. There is a tuberculous osteitis of the Haversian canals, and that process is a sort of unnatural bony regeneration. This is not the only case I have ever had in which bone regeneration has taken place. I have found a thick mass of connective tissue or of scar tissue in some cases.

Friedlander's bacillus was present in three of my cases that I examined. A large number of smears were examined as a matter of routine in my work.

Dr. Stucky: Did you have any trouble with those cases?

Dr. Beck: They are troublesome.

Another pathologic condition in which the ear is involved has not been mentioned, and that is edema associated with nephritis. This is certainly an affection which is present. There is edema of the mucosa of the middle ear.

Dr. Derrick T. Vail: Not to prolong the discussion too much, I will say a few words in regard to certain phases of this

subject. It has been mentioned by the essayists in these excellent papers that in some forms of otitis media the secretion in the ear is sterile. We know that to be the case. These cases frequently do not require paracentesis; they frequently clear up without operation by the let-alone treatment. Sometimes we have to do paracentesis of the drum, and we find a small amount of serum or mucus, which when let out is followed by a speedy recovery. If the surgeon himself has infection of one kind or another in his upper air passages and is handling a case of that kind, it is possible that he may or can infect the case with some of his own bacilli which are present in his breath and may be present on his fingers or on the instruments which he is handling. It is important, indeed, that this point should be recognized in handling these cases. In all cases of a surgical nature, this point should be borne in mind. Surgeons who have atrophic rhinitis have no right to operate on any case, I hold, without taking very strenuous precautions, that the patient be not infected with breath which is laden with bacilli. The person who is about to operate on a case of otitis of any kind should guard against producing infection quite as much as is now done by the operator in eye surgery, who not only sterilizes his hands and instruments thoroughly, but likewise wears a mask over his face and sees that every point is carried out and the patient is not infected. The promiscuous treating of cases out of the same cotton jar, with the same instruments, without sterilization, without washing of the hands, is almost criminal. The patients should be approached with the sacred feeling that they have already a disease without running the imminent risk of infection at the hands of the surgeon. I simply sound this note of warning, so that the point I have brought out may be fully appreciated. Much harm may be done through carelessness or indifference, and I have no doubt some cases are infected through the carelessness of surgeons themselves.

Dr. George F. Keiper—As to the etiology of the disease, it is rather hard to demonstrate the bacillus of influenza in the discharge from the ear, and yet we see cases of mastoiditis following influenza, which, as Dr. Stucky has said, are quite severe, there being an extension of the pathological process. I believe this extension will hold through with reference to cases due to scarlatina. We have an

infection, as it were, by direct continuity of tissue up through the Eustachian tube, and that seems to be the way in which infection occurs. I do not believe we have demonstrated the presence of the influenza bacillus, although we have looked for it carefully in our examinations in several cases.

With reference to the regeneration which takes place, it is our duty, as I pointed out in the beginning of the trouble, to conserve as much of the periosteum as we can. It is impossible to cover all surface with the periosteum we have left. It will retract. We can do some good with it and prevent a great deal of depression back of the ear, and it is our duty to look carefully after the periosteum in mastoid work.

Dr. Sauer (closing the discussion): I only wish to add a few words in regard to the remarks made by Dr. Stucky. He misunderstood me in regard to the percentage of primary tuberculosis. In my paper I said it is believed by Ismer that 13 per cent of all mastoid cases in children are tubercular, and that half of those are primary.

With reference to the statement made by Dr. Ballenger as to the frequency of suppuration in sinus disease, I will say that I did not go into that phase of the subject because it would have carried us too far. I mentioned inflammatory processes without going into details.

PAPER:

VACCINE THERAPY IN SOME SUPPURATIONS OF THE NOSE AND EAR; ALSO TECHNIC FOR DETERMINING OPSONIC INDICES.

JOSEPH C. BECK, M. D.

THE TECHNIC OF DETERMINING OPSONIC INDICES.

The facts and theories upon which opsonic treatment is based are as follows:

1. Bacteria infecting the body are attacked by leucocytes which ingest them.

2. The number of bacteria which can be ingested is of varying quantity.

3. The number of bacteria which can be ingested depends upon their preparation by substances present in the plasma of the blood, known as opsonins.

The exact nature of opsonins is not known, but it is known that they are not identical with the agglutinins, antitoxins, etc., which are also found in the plasma. Their action is not on the leucocytes, but on the bacteria which, as stated, they prepare for ingestion. Opsonins are present in normal blood as well as in the blood of infected individuals. The opsonic strength of normal blood is practically constant, but varies slightly with the individual, and general health, nutrition, etc.

The opsonic strength of an infected individual is lower than that of a normal individual, consequently his blood can prepare fewer bacteria and fewer will be ingested by the leucocytes. The relation between the number of bacteria ingested by the leucocytes of the infected person to the number of bacteria ingested by the leucocytes of a healthy individual, gives us a value which we call "the opsonic index." For instance, if ten bacteria are ingested on the average by the leucocytes of a healthy person, and five of the same bacteria

by the leucocytes from an infected person, the opsonic index of the latter would be .5.

The opsonic index of an infected person may be increased by injecting into him killed cultures of his infecting organisms. For instance, if an infection is due to the staphylococcus albus, some of these particular germs are taken, grown on suitable culture media, and when sufficient quantity has been obtained, the culture is washed off with .85 per cent sodium chloride solution, to which a little carbolic acid has been added. The mixture, well shaken, is standardized to contain 300,000,000 cocci to the cubic cm., which represents one hypodermic dose. It is important to inject cultures made from the particular infecting organisms, because it is well known that even such well known organisms as the staphylococcus albus are subject to greater variation in virulence. Furthermore, this prevents errors in the diagnosis of the infecting germs.

I. METHOD OF DETERMINING OPSONIC INDICES.

In a small test-tube, place about two cubic cm. of a solution containing 1½ per cent of sodium citrate, and .85 per cent sodium chloride. Clean the finger with soap and water, followed by alcohol; prick the finger, and allow fifteen drops of blood to flow into the solution in the test-tube, mixing well. The sodium citrate prevents coagulation of the blood. The test-tube is now taken to the centrifuge and centrifuged until all the solid particles of the blood are thrown down. The red blood corpuscles being the heavier, will form the lower layer, the leucocytes appearing as delicate, grayish film (known as "cream") on the surface of the reds. The supernated liquid has dissolved in it the plasma of the blood and is withdrawn by means of a delicate pipette, care being taken not to disturb the cream. The test-tube is then filled with .85 per cent sodium chloride solution, the corpuscles well shaken up, and the mixture again centrifuged. The corpuscles will then arrange themselves as before, and after withdrawing the supernated liquid, we will have freed them from the original plasma and the sodium citrate that was previously added. By means of special tubes, known as capsules, a quantity of blood is collected from the patient (capsule half-filled). Capsules of blood are similarly collected from several healthy individuals (two or three). The capsules are placed in the incubator at 37° C. for about five minutes, when the blood will be found to have coagulated. The capsules are then centrifuged, the

clot being thrown down, leaving the clear serum above. Meanwhile a suspension of the particular bacteria has been made by taking a loopful from the culture and mixing thoroughly with a quantity of .85 per cent salt solution, the amount of the solution being such that the mixture will be turbid, the turbidity to be judged only by experience.

We now have prepared: (a) the cream consisting of washed leococytes; (b) a suspension of the infecting bacteria; (c) serum from patients; (d) serum from several normal individuals.

We now have prepared: (a) the cream consisting of washed leu- of about 5 millimeters from its tip, we make a mark with a blue pencil. We now draw from the cream sufficient to fill the stem up to the blue pencil mark. Then we remove the pipette from the cream, drawing the volume a little way up to the stem so as to leave an air space. Then dip the same pipette into the suspension of bacteria of which we draw up an equal quantity. We leave another air space and draw up an equal quantity of serum from the patients' capsule. We now blow the contents of the pipette upon a glass plate and mix thoroughly by drawing up and blowing out several times. After mixing, the mixture is drawn well up into the stem of the pipette, the tip of the pipette sealed in a flame, the pipette marked on its bulb for identification and laid aside. We now take equal quantities of the sera from the capsules of blood collected from the normal individuals and mix them together in a little test-tube. This mixed serum is called "the pool." The object of mixing several sera rather than relying on one serum is to overcome fluctuations which are present in normal sera (fluctuating opsonic strength.) Another long-stemmed pipette is now taken, a distance marked off from its point as before, and equal quantities of cream, bacterial suspension, and pool serum taken and mixed, drawn up into the pipette, the end of the pipette sealed, its bulb marked for identification, and laid aside.

It will be noticed that these two pipettes vary only in that one contains patient's serum in the mixture, while the other is made up with pool serum. The two pipettes are now taken, placed in the incubator at 37° C. for fifteen minutes. They are then removed from the incubator, the contents of each is separately blown on a glass plate, drawn back and forth several times to insure mixture (this second mixing is required because the corpuscles tend to settle during incubation), and now a drop of the mixture is blown onto the

end of a perfectly clean glass microscopical slide, and the drop spreads evenly over the slide. At least three slides (two to be held in reserve for accidents) should be made from each pipette. The slides are now stained with Nocht's stain and examined with the microscope. The number of bacteria in each of fifty leucocytes is counted on each side. As before stated, the relation between the number of bacteria ingested by the leucocytes on the patient's slides compared with the number ingested by leucocytes obtained from the control slides, gives us the opsonic index. As will be noticed, the serum was the only variable factor in the preparation of these slides, therefore the variation in the number of bacteria ingested must be due to this difference of sera, and the substance in the sera which is responsible for this variation is "opsonin."

II. PREPARATION OF VACCINE.

A culture of the specific germs is washed off with salt solution and carbolic acid as before stated, standardized, placed in homeopathic phial (number for staphylococci being 300,000,000, other organisms requiring a strength of from 50,000,000 up) subjected to a temperature of 60° C. for a half to one hour in order to kill the organisms, a higher temperature to be avoided so as not to interfere with the chemical action of the bacterial ferments.

A control culture is made from the vaccine, and in certain cases also guinea pig inoculations to insure sterility of the vaccine; especially the absence of tetanus must be insured.

REPORT OF CASES.

The total number of cases that I have treated by this method is eleven, which are as follows:

2 Unilateral sinusitis of the staphylococcus variety of infection, of a chronic type.

1 Polysinusitis, chronic, of a staphylococcus aureus, pseudo-diphtheria bacillus.

1 Chronic antral infection, unilateral of dental origin, staphylococcus infection.

2 Subacute unilateral sinusitis (most probably confined to the frontal sinus). Staphylococcus pyogenes aureus.

2 Chronic purulent otitis media of a double infection; viz., staphylococcus and pneumococcus (bilateral).

1 Chronic purulent otitis, radical mastoid, Friedlander bacillus infection.

1 Chronic purulent otitis, unilateral, ossiculectomy, diphtheria infection.

1 Acute otitis media purulenta bilateralis diphtheria-like bacillus infection, following a violent grip attack.

All these cases had, previously to the vaccination treatment (autogonous), the usual accepted method of treatment without the desired results. All of them were subjected to the opsonic index which was lower than the normal; one as low as 0.3 and the nearest to the normal was 0.76. In all but one case, and that was the chronic bilateral sinus infection in which more than one index was taken, I depended almost exclusively on the clinical manifestations as an index and used the average time of ten days between the times of vaccination. Complete records were kept on the observations such as reaction and other symptoms and will be published in detail in a subsequent paper. Suffice it to say at this time that without exception there is a distinct improvement and some of the cases are cured, although not long enough time has elapsed in the chronic cases to be absolutely certain. My purpose in bringing this subject before you is not to come with any positive conclusions, because in the first place it requires a great number of cases of various pathological conditions and a much longer time to observe before one can make positive statements, but, to encourage if I can, some of you to take up this line of work if you are not already doing it, and in a symposium, report the results. So far as complications or accidents are concerned in this treatment, I must say that not in one case was there a single bad result and only one that gave me any thought and anxiety, and that was the following:

In the case of subacute unilateral sinus disease of staphylococcus pyogenes aureus infection, at the second injection into his left arm, there followed what clinically one would diagnose as an erysipelas with marked infiltration from the point of inoculation above the elbow, down to the finger tips, but absolutely no general symptoms. The patient felt as well as he ever did, and there was no infection of the axillary glands. Most of the cases showed slight general disturbances for from an hour to four hours after the vaccination, such as nausea, malaise and slight headache, and occasionally a slight rise of temperature.

The specific technic of vaccination is as follows: Scrub up thoroughly the part where the injection is made, either the arm or interscapular region, and under strictly aseptic precautions, draw up into

a hypodermic syringe the dose prescribed, usually one cubic centimeter of the vaccine, and inject subcutaneously, seal up the puncture with collodion. As said above, about ten days are allowed to elapse, when a second vaccination is made and so often repeated in those intervals until the patient is cured. If one should observe that a patient reacts badly and becomes markedly depressed after the injection, it is necessary to prolong the interval.

In conclusion, I wish to say that at the present time I have the following cases under treatment, but not sufficient length of time has elapsed for me to even report any partial results. Three cases of chronic nasopharyngitis, all staphylococcus infection (mild).

Two cases chronic tubercular laryngitis (ulcerated).

Four chronic purulent otitis media bilateral, in tubercular individuals, in which a positive ophthalmic reaction was obtained.

Two cases of atrophic rhinitis in tubercular subjects, one in non tubercular subject.

All these tubercular patients are treated by tuberculin injections instead of the autovaccine.

The laboratory work of determining opsonic indices and making the vaccines are at present done for me by the Columbus Medical Laboratory and the Chicago Laboratory.

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DISCUSSION.

Dr. M. A. Goldstein.—Dr. Beck, with his usual progressiveness and foresight, has started a new ball rolling, and I think it is up to us to develop the question of opsonins and opsonic therapy as rapidly as we can. It has been taken up in other departments of medicine more or less successfully, but very little of this work has been done in otology and laryngology, and I am in hearty accord with Dr. Beck that this work should receive some recognition from our department in this field, and I hope it will be within the province of some of us to develop this field of work. I think it is a special development in the infant bacteriology for a practical purpose.

Dr. Frank Allport.—We have been using the vaccine therapy in our office, but I have only had one case in which I have used it myself, and I got a most decided result with staphylococcus infection in that the discharge from the ear stopped in thirty six hours, and has never returned. This was about two months ago.

Dr. Brawley has used it in a dozen cases of infection of the accessory sinuses, and is very much pleased with the results so far. We are working along these lines in our office, and so far we have been gratified with the results. That is all I can say in regard to it.

PAPER:

DIAGNOSIS AND TREATMENT OF SUPPURATION OF THE LABYRINTH.

R. BISHOP CANFIELD, M. D.

The diagnosis of suppuration of the labyrinth is made from the symptoms evinced by the patient before operation and by the pathological condition discovered during operation.

Symptoms: The invasion of the labyrinth by a suppurative process in the middle ear is marked by no symptoms in the majority of cases. This is due to the fact that it occurs generally during a chronic suppurative otitis media associated with caries or cholesteatoma. Extension takes place slowly by a process of erosion from without. This allows the labyrinth apparatus time during which to accomodate itself to the gradual change in pressure and condition that occurs with the localized destruction of its bony capsule. It is the sudden invasion of the labyrinth or the sudden extension of disease within a labyrinth already entered that gives rise to symptoms. When this occurs it causes certain manifestations that are of diagnostic value. Of these:

Deafness is of importance. Unilateral deafness, as Bezold has pointed out, is difficult of diagnosis and consequently of doubtful value. When however a patient suffering from a suppurative otitis media develops under observation a sudden increase in deafness in the suspected ear the symptom is of great significance. The absence of deafness does not necessarily mean that extensive pathological change has not already taken place in the labyrinth for the vestibule may be filled with cholesteatoma and pus, and the cochlea extensively affected without materially affecting the hearing.

Disturbances of Equilibrium are frequently present. When standing, the patient often rests most of his weight upon the foot of the affected side. While walking he tends to turn off or bend toward affected side.

These disturbances are intensified if the eyes are closed. In some cases they are slight, but continuous, while in others they alternate with periods during which the patient is free of all inconvenience. In severe cases they are so marked as to prevent the patient from assuming the upright position or moving about without support. After being present for some length of time they have been known to disappear for years. Vertigo is common and is intensified by exertion. It varies greatly in degree and character. In some cases it is not more than dizziness, transient or continuous. In others typical and severe rotary vertigo appears. In one of my cases pressure upon the right semi-circular canal after operation caused all objects to rotate very rapidly to the right. Removal of pressure caused them all to snap back through the reverse arc quickly, until they reached their proper position. Pressure upon the posterior semi-circular canal caused the bed to slip suddenly away toward the foot. Removal of pressure brought it quickly back again. These sensations were so intense that the patient would hold tightly to the bed to keep from throwing himself on to the floor. In cases in which vertigo is most marked, nausea and vomiting are generally noted. They occur upon arising or follow exertion. Lateral nystagmus is less common. It rarely occurs without vertigo, and is most marked when the eyes are turned towards the healthy side. Fever and headache are rather uncommon, and are not at all characteristic.

The hearing test is of questionable value. The hearing for the voice in my own cases varied from the whisper at three feet to a degree which made it probable that the loud conversational voice was heard in the other ear, although that ear was hermetically sealed. The fact that a degree of deafness often occurs in chronic suppurative otitis media without extension of the infection to the labyrinth, and that extension into the labyrinth sometimes takes place without causing marked deafness, lowers the value of the voice test considerably, even when taken into consideration with other tests, and renders it worthless when considered alone.

The Weber is decidedly unreliable. It was not localized in several of my cases, and in others was localized in the suspected ear quite as frequently as in the other.

The Rinné was negative more often than it was positive.

The high limit, however, was always somewhat lowered, in one case was 512. In this case the Weber was not localized. The

lower limit is, of course, elevated on account of the middle ear involvement.

The symptoms mentioned are important in that they point toward the presence of an intracranial complication, but are not sufficient in themselves to render a diagnosis of labyrinth disease positive. They may all be present in cases in which the labyrinth is not involved. This is demonstrated by the following case:

Mrs. S., 30. years. Scarlet fever at age of 4 years. Both ears were affected. Right ear soon ceased to discharge, but left has continued to discharge at times ever since. At time of first discharge had complete left facial paralysis. This lasted for about two months. Since then has had times when paralysis was apparent, lasting several weeks. Since first attack paralysis has never been complete. About two weeks ago, during a head cold, was taken with severe rotary vertigo, ringing tinnitus and increase in deafness in the left ear, pain in the left occipital region, slight foul discharge from left ear. Temperature has reached as high as 105° with frequent remissions, no chills, yesterday delirium, pupils dilated, breathing difficult, apparently in extremis. To-day remarkable change for the better.

Examination.—Patient well developed, intelligent woman, perfectly rational, but inattentive, complaining of pain in region of left ear, high pitched tinnitus left ear, temperature 101°, pulse 80, respiration 20, leucocytosis 14,000. Knee jerks slightly exaggerated. Right ear shows evidence of old suppuration, but no sign of acute inflammation. Left ear: M. T. present, retracted, scar in superior half, small perforation in postero-superior quadrant contains somewhat tenacious pus, probe passes into attic. No drooping of postero-superior canal wall, no mastoid tenderness. Hearing test somewhat unsatisfactory on account of patient's condition.

Right.		*Left.*
Loud conversation	Voice	Loud conversation.
Not localized	Weber	Not localized.
Negative	Rinné	Negative.
256 Slightly, Higher fks. 0	A. C.	256 slightly, Higher fks. 0.
Apparently somewhat decreased.	B. C.	Apparently somewhat decr.
Dench 3	High	Dench 4.
256	Low	256.

Paracentesis permitted escape of foul pus and cholesteatoma. Diagnosis of chronic suppurative otitis media, cholesteatoma and probable involvement of the labyrinth was made and radical mastoid operation advised with exploration of labyrinth if indicated at time of operation. Operation was refused. Patient remained in same condition for two days and then had another attack of pain, increase in stupor, accession of temperature to 105° without chill, increase in deafness, tinnitus and vertigo from which she made a slow recovery.

A radical mastoid operation was performed. Tympanum and mastoid found filled with cholesteatoma but the labyrinth was not involved. Uneventful recovery. It is interesting to note that after the radical operation the upper limit became normal and hearing for conversational voice increased to six feet.

The labyrinth symptoms and hearing test indicative of labyrinth involvement were no doubt due to pressure upon the labyrinth of the cholesteatoma and to incident congestion.

Even more important than the fact that symptoms indicative of invasion of the labyrinth can be present during the course of a chronic suppurative otitis media without disease of the labyrinth is the fact that invasion of the labyrinth can take place without the presence of a single symptom on account of which the condition may be suspected. Of my own cases marked deafness was present in 3, disturbances of equilibrium in 5, vertigo in 5, nausea and vomiting in 3, nystagmus in 2, no symptoms whatever in 5.

The diagnosis of labyrinth suppuration can at present be made with certainty, in the great majority of cases, at least, only at the time of the radical mastoid operation, by discovery of disease of the inner tympanic or mastoid wall and of a sinus leading into the interior of the labyrinth. These sinuses appear in order of their frequence in the semi-circular canals, through the oval window, through the promontory, and through the round window. Perforation into the horizontal semicircular canal is frequently easy of diagnosis. This is true when the perforation occupies a point just below the most prominent portion of the semicircular canal. In such cases it appears as a single opening into the lumen of the canal, or, if more destruction has taken place, as two openings one leading into each branch of the canal. I have seen several such perforations without signs of caries, granulation tissue or pus within the canal. The diagnosis is more difficult although the destruction

is greater in cases in which the posterior half of the horizontal semicircular canal is lacking. With the perforation in this position the operator may be left in doubt as to its existence despite perfect illumination and painstaking investigation. Pus is rarely seen issuing from such a perforation.

I have seen it in but one case. In that case the vestibule was filled with fluid pus. More frequently a small bud of granulation tissue is noted within the canal. When this is wiped away the observer may be in doubt as to whether the interior of the canal is involved or not, possibly concluding that the granulation tissue is due entirely to the disease within the antrum. I believe that such indecision is justified and that perforation may occur in the semicircular canal, exist for a time, become closed by granulation tissue and later by bone, with but slight, if any involvement of the labyrinth. In one case in shaving down some cancellous bone over the horizontal semicircular canal in a patient in whom no labyrinth symptoms had been noted I cut away a small irregular mass of bone the removal of which opened the canal. This I think was new bone formed in the successful attempt on the part of nature to close a perforation and protect a labyrinth from invasion. The posterior semicircular canal is somewhat more deeply situated than the horizontal and is more deeply covered with cancellous bone, consequently its exact delineation is somewhat more difficult. On this account perforation through the canal is sometimes missed. When the canal is thoroughly exposed the perforation is easily seen occupying the most prominent portion of the canal. It is sometimes seen on the anterior surface and associated with destruction of the posterior branch of the horizontal canal. In some cases very small pneumatic cells may cover the canal and be especially numerous in the neighborhood of the solid angle. They sometimes extend rather deeply along the walls of the canals and if filled with freely bleeding granulation may be mistaken for a perforation. In this connection the systematic use of the fine probe is of great diagnostic value. This instrument must be employed with judgment. It is well recognized that in cases which exhibit a single perforation in this neighborhood the labyrinth disease is frequently definitely localized and that this localizing process takes place by the formation of a wall of protecting granulation at the deepest limit of the disease. It is without question highly dangerous to pass a probe further into the semicircular canal

than this protecting wall. Such a procedure only carries infection into the deeper portions of the labyrinth and disseminates the disease. It does not afford the observer any additional information. As the point at which this protecting wall has formed can not be told, the probe is to be passed only deeply enough to render the diagnosis of perforation positive.

It is generally stated that next in frequence to the neighborhood of the semicircular canals perforation takes place through the oval window. In the presence of the stapes this perforation must be difficult of diagnosis. In the absence of the stapes perforations can sometimes be made out, if not by infection, then by means of the fine probe. Hinsberg states that infection can pass through the oval window in cases in which macroscopical change in the window can not be discovered. In support of this statement I have been able to demonstrate caries of the vestibule in a case in which I could not discover a perforation. The absence of the stapes is not always significant of perforation through the oval window as it was noted in one of my cases in which no perforation could be detected and in which fair conversational voice was present after operation. I have seen perforation of the promontory in one case in which about two-thirds of the promontory was lacking. Cases have been reported in which infection reached the labyrinth from the cranial cavity through the internal auditory meatus or the aqueducts. I am inclined to think that the internal auditory canal was the avenue of infection in one of my cases.

Much difference of opinion exists concerning the pathological significance of perforations in various positions of the external labyrinthine wall.

In my cases, judging from hearing test and result of operation, single perforations into the horizontal semicircular canal occurred only in cases of circumscribed labyrinthine disease. Double perforations, one in the horizontal and one in the posterior semicircular canal, occurred in two cases in which the vestibule was filled with pus and cholesteatoma. It would seem that double or multiple perforations must always speak for an extensive pathological condition. In considering the value of visible perforations in the diagnosis of labyrinth suppuration secondary to suppurative otitis media it must be remembered that the disease has been demonstrated clinically and at autopsy in cases in which perforation could not be recognized.

The indications for operation upon the labyrinth are met in two classes of cases.

1. Those in which symptoms referable to labyrinth disease have not been present before operation.

(a) When a single perforation is discovered through a semicircular canal, from which pus escapes.

(b) When a perforation through the oval window or promontory is discovered, whether pus is seen or not. In these cases marked deafness is present and extensive disease of the vestibule must be suspected.

(c) When labyrinth symptoms develop suddenly after a radical mastoid operation. The majority of these cases are due to a latent labyrinth suppuration an acute exacerbation of which has been set up by the operation. In such cases the vestibule is to be opened widely and further operative measures governed by its condition. In isolated perforations through the horizontal semicircular canal in which other evidence of disease can not be demonstrated, operation upon the labyrinth may be postponed.

This is in support of Hinsberg's statement that, in his experience, of all cases of circumscribed semicircular canal disease not a single patient succumbed to the consequences of the labyrinth suppuration. In this connection he points out the necessity of watching very carefully cases in which a single perforation of a semicircular canal has been demonstrated so that on the appearance of vertigo, nystagmus and diminution of hearing, the labyrinth may be promptly opened.

2. Those in which labyrinth symptoms have been present before operation.

(a) When no fistula is found.

(b) When fistula is found.

When no fistula is found. This class of cases, it seems to me, should be very small and should include those cases which have developed labyrinth symptoms while under observation and those in which deafness typical of disease of the perception apparatus can be demonstrated. Symptoms highly suggestive of labyrinth suppuration so frequently disappear after the radical mastoid operation that unless the pathological change in the tympanum and mastoid is altogether too slight to account for the labyrinth symptoms delay may be allowed. To this class belong also those cases in which a deep seated cerebellar abscess or extradural abscess is suspected, (Jansen.)

(c) When a perforation into the labyrinth is found in a case that has previously shown labyrinth symptoms, the indication for opening the labyrinth is clear.

The following six cases are illustrative of the fact that invasion of the labyrinth can take place without symptoms, while the next five show some symptoms characteristic of the condition.

8-17-05. *Case* 1. Percy B., 15 years. Discharge from left ear following scarlet fever 11 years ago. Discharge has persisted with frequent remissions ever since. No symptoms except moderate deafness and foul discharge.

Examination: Right ear negative. Left: Great destruction of M. T. Tympanum filled with granulation, pus and cholesteatoma. No mastoid symptoms. Large amount of adenoids.

Right.	*Hearing test.*	*Left.*
Loud conv. voice.	Voice.	20/21
+	Weber.	
—	Rinné	+
Dench 3	High.	N
512	Low.	N

Radical mastoid operation discovered tympanum filled with cholesteatoma, antrum containing moderate amount of granulation tissue, pus and cholesteatoma. Malleus and incus wanting. Small sinus leading into posterior branch of horizontal semicircular canal. No evidence of pus in canal, no caries, Koerner flap and skin grafts later. Sinus moderately enlarged for purpose of examination. Adenoid operation. Slight vertigo after operation.

5-3-06. *Case* 2. Ivan P., 10 years. Chronic suppurative otitis media since scarlet fever seven years ago. No symptoms except moderate deafness and discharge.

Examination: Right ear. Great destruction of M. T. Ossicles not seen. Tympanum contains granulation, pus and cholesteatoma. Probe passes into attic and discovers caries on external attic wall and in direction of antrum. Left ear: Scars in M. T. No evidence of present suppuration.

Right.	*Hearing test.*	*Left.*
L. C. V. at ear.	Voice.	15/21
+	Weber.	
—	Rinné	+
D3	High.	N
512	Low.	256

Right radical mastoid operation. Cholesteatoma in tympanum, antrum, and mastoid, peri-sinus collection of cholesteatoma. Perforation in horizontal semicircular canal leads into vestibule. This opening enlarged. Thiersch grafts in Eustachian tube and tympanum, Koerner flap. Initial incision sutured. Recovery without marked labyrinth symptoms.

7-8-07. *Case* 3. Fred H., 17 years. Discharge from right ear for 15 years without symptoms.

Examination: Right ear. Polyp occludes canal. From around polyp escapes foul cholesteatoma. Sinus through posterior canal wall passes into mastoid.

Right.	*Hearing test.*	*Left.*
Loud conv. voice.	Voice.	20/21

Radical mastoid operation. Tympanum, antrum and mastoid filled with true cholesteatoma. Dura over middle cerebral fossa and over sinus laid bare by the disease. Polyp attached to inner wall of antrum. Sinus through horizontal semicircular canal leads into vestibule. Sinus enlarged. Koerner flap. Initial incision sutured. Moderate vertigo for several days after operation. Discharged well.

11-21-07. *Case* 4. Madge S., 9 years Recurrent discharge for the past three years, continuous from the left ear for the past year. No history of labyrinth symptoms.

Examination: M. T. shows great destruction. Tympanum contains cholesteatoma and pus. Ossicles not visible. No mastoid tenderness. After several weeks' treatment without avail, radical mastoid operation. Most of the disease in and in the vicinity of the antrum which contained cholesteatoma and caries. Fallopian canal lacking for about 1-4 inch in the immediate vicinity of the horizontal semicircular canal. Horizontal semicircular canal shows almost complete destruction, remainder of canal carious. Dura and sinus laid bare. Short Koerner flap. Initial incision sutured. Skin graft later. Complete recovery without labyrinth symptoms.

12-30-05. *Case* 5. Frances McD., 18 years. Continuous discharge from both ears since measles 13 years ago. Severe headache but no other symptoms.

Examination: Both ears showed marked destruction of M. T. Tympanum contains cholesteatoma and pus. Caries in both attics. Left shows considerable destruction of posterior canal wall.

Hearing test not taken.

1-11-06. Radical mastoid operation left side. Extensive sclerosis. All of posterior semicircular canal and posterior half of horizontal semicircular canal found destroyed. Opening enlarged. Koerner flap. Thiersch grafts to tube and tympanum. Moderate vertigo after operation.

Stacke operation on the right side at a later date. Labyrinth not involved.

8-28-05. *Case* 6. Cleo F., 15 years. Both ears have discharged since the age of 6 months. No nausea or dizziness or other labyrinth symptoms.

Examination: Both ears show canals filled with granulations, pus and cholesteatoma. Sinus leads through the posterior canal wall of the left ear into mastoid. Adenoids and tonsils.

Right.	*Hearing test.*	*Left.*
L. C. V.	Voice.	L. C. V.

Radical mastoid operations on both ears at different dates. Right side showed moderate amount of cholesteatoma, granulation tissue, and pus throughout tympanum and mastoid. Labyrinth had been invaded through the posterior semicircular canal which had been completely destroyed. Horizontal semicircular canal also involved. Thiersch graft of tympanum. Initial incision sutured. Moderate labyrinth symptoms after operation. Mastoid operation followed by removal of adenoid.

Left radical mastoid operation showed extensive cholesteatoma formation with exposure of the facial nerve but no involvement of the labyrinth.

3-15-07. *Case* 7. Frank R., 14 years. Right ear has discharged for ten years, cause unknown. No symptoms until one month ago when complete facial paralysis and vertigo with increase in deafness. Patient says that at that time and for several days he staggered to right but did not fall.

Examination: Canal occluded in its middle third by drooping of superior canal wall which touches inferior. By inserting speculum beyond occluded area small amount of stale discharge and glistening polypi are seen. Complete facial paralysis. No mastoid tenderness. Adenoids and tonsils.

Radical mastoid operation. Tympanum, antrum and mastoid filled with foul pus. Fallopian canal shows perforation about 1-4 inch

long, downward from level of horizontal semicircular canal which shows a perforation that uncaps canal and shows openings into both branches. Openings enlarged. Koerner flap. Initial incision sutured. Insignificant vertigo after operation. Adenoid and tonsil operation at a later date. Complete recovery from facial paralysis in three months.

5-13-06. *Case* 8. Wm. G. H., 22 years. Right ear has discharged since measles, 17 years ago, with occasional exacerbations. Polyps removed from both ears one year ago. Two months ago while undressing had his first attack of vertigo which was so severe that he fell over onto the bed. This vertigo has continued with periods of remission and exacerbation up to the present time. No nausea or vomiting. Acknowledges syphilis several years ago. No recent symptoms.

Examination: Patient sits with head bent to right. Not interested in surroundings. Answers question slowly but correctly. Apparently not in pain. Pupils equal, react normally, no nystagmus, no strabismus. Disturbance in equilibrium marked upon arising, stands with most of his weight upon the right foot, while walking turns off and bends to the right.

Ears: *Right* canal contains creamy pus. Depths of canal filled with easily bleeding granulations. Drooping of superior wall of the canal just external to the tympanum. No mastoid tenderness. *Left.* Dry, depths are filled with bloody crusts.

Right.	*Hearing test.*	*Left.*
1/12/35	Voice	2/35
Not localized.	Weber	Not localized.
512	High.	N
	Low	512

Radical mastoid operation right side. Marked superficial sclerosis of mastoid. Deeper parts filled with cholesteatoma. Horizontal semicircular canal completely destroyed. Granulations in tympanum. Koerner flap sewed to posterior wall of concha. Initial incision not sutured. Skin grafts at a later date. For several days severe rotary vertigo to the right. This gradually wore off. While walking the patient had the inclination to turn off to the right but was able to walk in a straight line with some effort. Three months after operation had two attacks of severe vertigo with some nausea. Examination revealed complete epidermiza-

tion of the operative field. No apparent trouble. Since then no report of patient.

8-5-05. *Case* 9. Patty Van T., 9 years. Has had continuous discharge from both ears since scarlet fever six years ago. Two weeks ago severe pain right side with decrease in amount of discharge, post-aural swelling on the third day. Since attack patient has been very dizzy but has been too sick to complain.

Examination: Patient evidently very ill. Temperature 103°. Pulse 110, very weak and thready, sepsis, adenoids. *Right Ear*: Canal occluded by drooping of superior wall. Large post-aural swelling extending to cheek. Cholesteatoma in canal. *Left Ear*: M. T. gone. Tympanum filled with cholesteatoma. Right radical operation with curettage of vestibule. Cortex of paper thinness. Five perforations externally. Tympanum, antrum and mastoid filled with cholesteatoma. Two perforations into middle cerebral fossa. Pus and cholesteatoma on sigmoid sinus. Posterior semicircular canal and posterior half of horizontal semicircular canal destroyed. Vestibule filled with pus. Left radical mastoid at later day. Similar condition except labyrinth not involved. Discharged well.

10-8-06. *Case* 10. Mrs. Wm. J. B., 42 years. Patient had scarlet fever at age of five years. Both ears discharged for several years thereafter. Later the right ceased but the left has troubled her until the present time. Slight facial paralysis. For about a year she has had frequent and severe sick headaches, with nausea, vomiting and rotary vertigo. Three weeks ago during a severe headache she suddenly became unconscious. This lasted for several minutes after which she was helped to bed. Since then she has had continuous headaches over the entire head. Patient is two months pregnant but thinks that that has nothing to do with her symptoms, judging from previous experience.

Examination: *Right ear* shows great destruction of M. T., tympanum dry. *Left ear.* Two perforations in posterior half. Moderate amount of discharge having the odor of cholesteatoma.

Right.	*Hearing test.*	*Left.*
L. C. V.	Voice.	L. C. V.
Not localized.	Weber.	Not localized.
n.	High.	15 D
	Low.	1024

Patient is very unsteady on her feet, sways from right to left, has some difficulty in maintaining the upright position. When walking turns off to the left unless she is careful.

Radical mastoid operation. Mastoid almost completely sclerosed. No antrum discovered. The position of the antrum could be definitely made out but the cavity itself was filled with new bone. Tympanum filled with cholesteatoma. Promontory lacking in great portion, this causing a perforation in vestibule and first whorl of cochlea. This opening enlarged. Following operation vertigo was marked for ten days after which it gradually disappeared. Discharged well.

Case 11. Tiny B., schoolgirl, aged 14 years. Was brought to the University Hospital on account of a suppurative middle ear disease of the left side.

History: Patient had scarlet fever at the age of one year. Both ears discharged at that time. At the same time a swelling appeared behind the ear. This was lanced and the wound soon healed. The discharge from the ear, however, persisted for about two years. From that time a little discharge appeared at times. About a year ago, during a head cold, pain in the left ear reappeared together with an increase in the amount of discharge. Two weeks previous to admission to the hospital, during a head cold, pain reappeared again and discharge ceased. Two days previous to admission a swelling was seen over the mastoid. About the same time child became much weaker. She has had at no times chills, nausea or vomiting.

Examination: Shows a child fairly well nourished lying upon her right side, head markedly retracted, legs flexed on thighs, face flushed, eyes closed, mouth open, patellar reflexes absent, Kernig's symptom marked. The heart, while fairly strong when patient is lying down, becomes irregular and weak during exertion. She answers questions intelligently but slowly and sometimes requires several efforts before she is able to finish her sentence. She protrudes the tongue fully but with some hesitation and unsteadiness.

Temp. 102.8°, Pulse 140, Resp. 24, Leucocytes 17,000?

Local Ex. *Right ear*. Shows effect of a previous suppuration.

Left ear: External auditory canal filled with foul cholesteatoma and pus. Lumen of canal narrowed by pressure from mastoid. Over the lower half of the mastoid is present a smooth swelling,

very sensitive to pressure and showing slight fluctuation. This swelling extends beneath mastoid tip into the deep neck muscles.

A diagnosis of chronic suppurative middle ear disease with cholesteatoma and meningitis was made and a bad prognosis given. Careful questioning of the mother showed that the child had begun to complain of headache only the day before and that all her bad symptoms had come on suddenly. Therefore it might be supposed that her meningitis had begun within the recent few hours, had come on suddenly, was progressing rapidly and might at that time be confined to an area that could be reached by operative measures. The writer could recall five cases of purulent otitis meningitis operated upon successfully in his own practice and therefore felt warranted in suggesting an operation as the only possible means to offer even the slightest chance of recovery. The child was therefore immediately prepared for operation.

Under ether anesthesia the usual mastoid incision was continued down over the area of swelling in the neck and several drachms of foul pus and cholesteatoma were allowed to escape. Perforation through the inner surface of the mastoid tip had taken place on to the deep muscles of the neck. The mastoid cortex was completely removed and the mastoid cavity found of moderate size and filled with a true cholesteatomatous tumor having a well defined limiting membrane. This membrane had ruptured and allowed the escape of its fluid contents. Careful removal of the tumor showed the bony wall of the inner table destroyed by the disease so that the cerebellum was exposed over an area about the size of a silver dollar. Over this area the membrane of the cholesteatoma was firmly adherent to the cerebellar tissue, i. e., a true subdural cholesteatoma. The dura in the neighborhood was partly covered with foul purulent granulations. Those were removed and the brain uncovered until healthy dura was uncovered on all sides, except over a small area near the median line. The reason for not going further in the direction will be explained later. The lateral sinus was found uncovered for a distance of about an inch and a half but its lumen had not been invaded. The horizontal semicircular canal had been destroyed and the labyrinthine vestibule opened. A large amount of pus was secured from the posterior cerebral fossa. All pneumatic structure as far as possible was then removed including the mastoid tip and the posterior root of the zygoma and the wall of the external canal, thus converting the

middle ear, canal and mastoid into one cavity with smooth walls. The tympanic end of the Eustachian tube was then curetted in order to secure its future closure and a counter opening made in the neck for drainage. At this point, in spite of the hypodermic stimulation and of hot saline enemata repeated during operation the patient suddenly showed signs of complete collapse. Further operating was postponed and the patient returned to bed where she made a slow recovery from the anesthetic, regaining consciousness eight hours later.

9 P. M. Patient was perfectly conscious but restless.

She answered questions satisfactorily but slowly. Protrusion of the tongue showed paralysis of the left half of the tip. Opisthotonos became more marked with double optic papillitis.

5-10-07. She suddenly became comatose and died.

The autopsy: Brain showed slight tenseness of dura, considerable congestion of the pia, slight milky exudate over the left temporal lobe. Several drachms of plastic exudate over left cerebellar area corresponding to field of operation and extending inward and downward so as to involve structures of the base. True cholesteatoma membrane adherent to pia, left lateral ventricle filled with flaky fluid. Choreoid plexus congested. Cord shows several drachms of cloudy fluid in the arachnoid of the lower dorsal and lumbar cord. The facts that the cholesteatoma occupied a position in immediate relationship with the internal auditory canal, and that the changes in the vestibule were much greater than those in the semicircular canal system makes the inference possible that infection reached the labyrinth through the internal auditory canal, and that the perforation through the horizontal semicircular canal took place from within outward, at least examination of the bone makes it evident that the changes within the vestibule antedate those in the canal.

Treatment: In all cases in which labyrinthine suppuration has been suspected before operation or has been diagnosed at the time of operation by the discovery of a sinus leading into the labyrinth the radical operation must be performed. Not only is this the only operative procedure by which the suppurative process can be dealt with but it is the only one that gives a satisfactory view of that part of the labyrinth wall through which infection has penetrated.

It has already been pointed out that infection reaches the labyrinth through the region of the semicircular canal, the oval window, round window or the promontory. In order thoroughly to expose these areas the last steps in the radical operation must include the careful delineation of the three canals, the complete obliteration of the facial ridge even to the exposure of the facial nerve, the removal of the inner end of the anterior canal wall and of the external wall of the hypotympanum. The depths of the wound are then packed with gauze soaked in adrenalin in order that the field may be rendered bloodless and careful inspection of the labyrinth wall facilitated.

The complete exenteration of the labyrinth includes three distinct steps.

1. The entrance into the vestibule through the semicircular canal system.
2. The counter-opening through the oval window.
3. The operation upon the cochlea through the promontory.

The operative field is so contracted and the relationship between the vital structures so intimate that a perfect technique must be secured by work upon the cadaver before the exploration of the interior of the labyrinth can be attempted with safety to the patient or credit to the operator.

The entrance into the vestibule through the semicircular canal system is chosen as the first step as it offers a more comprehensive view of the vestibule than does the opening through the oval window, it is attended by less danger and less difficulty, and finally it sometimes reveals a localization of the pathological process that renders further operative interference unnecessary. Removal of the semicircular canal system follows its satisfactory delineation. It is conveniently begun by shaving down with a curette or sharp chisel the superior margin of the horizontal semicircular canal in its posterior half.

This is continued until the canal is well opened. In the same way the posterior canal is widely opened. Then by means of the mallet and sharp narrow chisel the superior wall of the horizontal canal is followed directly inward until the vestibule is reached. The posterior and superior canals are then obliterated. The chief danger during this step in the operation lies in injury to the facial nerve. This is avoided by not interfering with the inferior wall of the horizontal canal which is in most intimate relationship with

the Fallopian canal. I have demonstrated many times upon the cadaver that the chisel must under no circumstances be allowed to impinge upon the wall of the Fallopian canal, nor to rest upon the crest of the previously opened horizontal canal while this canal is being obliterated as the Fallopian canal will certainly be fractured. It is a wise precaution, I think, against injury to the facial to uncover the nerve for a considerable area of the horizontal portion in order that its exact position may be determined. Care is to be used in curetting the inner wall of the vestibule which is very thin, and rupture of which is attended by considerable danger, to both facial and auditory nerves. If painstaking examination seems to show the disease is limited to the semicircular canal system further operative measures may be postponed. If on the other hand the interior of the vestibule suggests further extension of the disease or if a sinus leading through the oval window or promontory has been discovered, the second step is undertaken. This consists in enlarging the oval window downward by means of a very sharp narrow chisel. In this way the posterior half of the inner tympanic wall is removed. With the greatest caution in order to avoid fracture of the inner wall of the vestibule into the internal auditory meatus, this opening is enlarged until satisfactory examination of the vestibule can be made.

The third step is the operation upon the cochlea. This, as J. D. Richards has recently pointed out, is the most dangerous step in the operation not only on the account of the most intimate relationship that the carotid artery, the jugular bulb, and the facial nerve bear to the field of operation but especially on account of the position of the very delicate and fragile modiolus that occupies the center of the cochlea. The modiolus is a hollow cone whose side forms the peripheral end of the internal auditory canal and whose apex is at a point an eighth of an inch in front of and on a level with the oval window. From his experiments and observations Richards concludes that the apex of the cone can be removed down to the first cochlear whorl without bringing the operative field and the intra-dural sac into gross communication and so rendering a fatal meningitis probable. Measurements of a number of temporal bones seem to prove to me that the internal auditory meatus terminates between 1-8 inch and 3-16 inch internal to the promontory. Consequently to do more than uncap the pyramid must be exceedingly dangerous and is rarely necessary. The cochlea is opened

safely by choosing a point 1-4 inch in front of the oval window and chiseling downwards and forward very gently until a cochlear whorl appears. This is enlarged backward to the vestibule and forward as far as possible, chiseling, as Richards advises, always in the direction of the long axis of the pyramid. In this way two cochlear whorls are widely opened. This is followed by the cautious removal of the remainder of the promontory which permits inspection of the entire upper whorls, and almost all of the lower whorl. My experience upon the cadaver leads me to believe that in all cases in which indications are not absolutely plain operative measures should cease at this point, although a small portion of the upper half of the lower whorl has not been seen. Even if caries at this point can be demonstrated it seems safer to allow this part of the cochlea to sequestre rather than to remove it with the probability of opening the internal auditory meatus. Further removal of the upper portion of the cochlea also endangers the support of the facial nerve in the labyrinth. In cases in which this portion of the cochlea is perfectly loose it may be removed. My experience with operations upon the cochlea is limited to two cases, one of my own in which I opened both whorls of the cochlea and removed the apex of the modiolus in a case in which caries was easily demonstrable, and one of Jansen's in which the cochlea was found to have sequestred and was completely and safely removed.

PRESENTATION OF CASES AND SPECIMENS.

Two Cases of Brain Abscess.

Dr. John F. Barnhill.—I wish to present a verbal report of two cases of brain abscess. It has been the aim of those of our local men here to present cases that were of unusual interest, or else something along the line of the papers that have been or are to be read. As you see, these two cases of brain abscess have recovered.

Case 1. This woman was operated on in 1902. She had an enormous brain abscess from an acute otitis media. Her case was reported in the *Annals of Laryngology, Rhinology and Otology* of that year. Her intelligence is perfect. Her health is better than it was before the operation, and there has been no untoward symptom of any kind since her recovery some six or eight weeks after the operation. The abscess was temporo-sphenoidal. This abscess was so large that when it was opened an examination of the interior of the skull gave me the impression that everything had escaped. The whole skull cavity seemed to be empty.

Case 2. This young man is from the Deaf and Dumb institution, and his case was one of acute temporo-sphenoidal abscess. In this instance we had a cerebral hernia which persisted for four or five weeks and gave considerable annoyance, and I am told that since his recovery he has had two petit mal attacks. He is a printer by occupation, at the institution. He works hard, and the superintendent tells me that he is one of the best employes there. Both these patients had a section of bone removed over the squamous portion of the temporal bone and a counter opening, together with a complete mastoid exenteration, such as we do in operations for acute mastoid disease. All of the cases that I have ever operated on for brain abscess, making a counter opening through the tegmen, have recovered; but all cases operated on without that counter opening have died. I believe the petit mal attacks that this young man has had are due to the scar tissue

which was the result of the cerebral hernia. The cerebral hernia was as large as my fist and for two weeks I expected the young man to die almost any day, and why he did not, I do not know.

Cyst of the Tonsil.

Dr. M. A. Goldstein: I want to present this specimen on account of the unusual size of the cyst, rather than because of its unusual character. It is a cyst of the tonsil. You have all seen such cases time and again, but I doubt whether any of you have seen any cyst of the tonsil larger than this. The patient was a medical student who was operated on three weeks ago. I preserved the cyst intact and found that it was contained within practically the original fibrous capsule of the tonsil. The entire capsule contains a cystic mass.

The pathology has not been thoroughly scrutinized, but it is evident the tonsil has been absorbed entirely in this cystic mass of tissue.

MEETING OF WESTERN SECTION, HELD IN CORONADO, CALIFORNIA, APRIL 21, 1908.

PAPER:

ACUTE OTITIS MEDIA IN INFANCY AND CHILDHOOD.

H. BERT ELLIS, M. D.

Diseases of children have been and still are neglected to a great extent, more neglected than any other branch of the practice of medicine. This is largely due to the fact that diagnosis is particularly difficult, the infant being unable, through lack of language, to give any idea of the physical symptoms; and the young child too imaginative to correctly express the condition.

In infants, it is not easy to diagnose otitis media, not only are the parts very small but the external meatus is apt to be filled with epidermis scales. In dead nurslings, purulent inflammation of the ear is found at autopsies in four-fifths of the cases; this form of trouble is usually latent, and in other cases, suggested by restlessness and tossing of the head. This concomitant otitis differs quite sharply from the ordinary form which has much more pronounced symptoms,—the most prominent of which is pain, but in the most destructive lesions of scarlet fever, pain is sometimes conspicuously absent, or at least not complained of by the apathetic child; this latter fact is of great importance, and must not be lost sight of by the general practitioner. On the other hand, severe symptoms simulating brain lesions, stupor or convulsions, may arise in simple otitis and these be immediately relieved by paracentesis, hence there is a need of examination of the ears in all such cases, for the little patients thus affected cannot tell of their earaches.

At birth, the inferior and exterior walls of the external meatus and the membrana tympani are in simple coaptation and the

irregularities filled with desquamated epithelium, the canal opens in a few days, forming an hour-glass canal (smaller at the inner end) the axis of the meatus being directed upward, causes the downward pulling of the auricle to inspect the membrane, which together with the cavity is about as extensive in the child as in the adult. A sieve-like bone separates the floor of the tympanum from the carotid canal and the jugular fossa. "The oblique position of the meatus and the thin plate of bone allow a puncture of the jugular bulb during paracentesis."

"The greater resistance of the drum membrane in the young, (the external cutis layer being often thicker than in the adult), the median connective tissue membrane very solid and the inner mucous membrane and its pavement epithelium at least as normal as in advanced age, contribute to the comparative infrequency of perforations." (Jacobi . . . Otitis Media in Children. Archives of Otology, Ap'l. '05.)

The relative infrequency of spontaneous perforation, and the anatomical difficulty of drainage, makes the purulent secretion in the tympanum seek other methods of escape. At birth, the temporal bone is loosely connected with the cranial bones, is vascular and traversed by many foramina containing blood vessels. The outer walls are more spongy than the tegmen, and hence the readiness of post-aural abscesses in infancy.

Owing to the influence of gravitation and the fact that the young periosteum and the numerous foramina are readily dissected, the direction of least resistance is outward usually,

(a) Upward and outward through the Rivinian fissure in the antral roof and backward between the bone and periosteum to form abscesses behind and above the ear;

(b) Through cartilaginous fissures, involving posterior auricle outward and forward in the characteristic way:

(c) Through the imperfectly closed roofs or the communicating blood vessels and lymph channels, directly to the middle fossa, setting up a meningitis or subdural or brain abscess:

(d) From the same sources, jugular thrombosis.

In the very young, because the sinus is a flat affair, it is not involved, but in older children, through carious softening or through the medium of intercommunicating structures, there may be thrombosis of the lateral sinus.

(e) The process may extend by way of the posterior surface to the cerebellum.

These pathological processes being so numerous, serious and likely to occur, there is a sharp demand for early, prompt, and if possible, prophylactic treatment. Immediate adenectomy on involvement, careful and extensive incision of the drum, are the early procedures.

Another important fact is that the lymphatics are nine times more permeable in infancy than in the adult.

The differences between the adult and the infant Eustachian tubes are briefly as follows:

1. The tube is much shorter at birth, less than one half as long (15 mm to 38 mm.)

2. The orifice and calibre are nearly as great as in the adult, hence proportionately much wider.

3. The child's tube is straight, having no angle at the isthmus.

4. The tube is nearly horizontal; in the adult, the pharyngeal end is 12 to 14 mm. lower.

5. The mouth of the tube in the pharynx, in a child at term, is on a level or slightly below the plane of the hard palate.

All these conditions favor the entrance of germs into the middle ear through the tube from the inspired air and nasal secretions.

The presence of vascular lymphoid tissue at the door of this open pathway (often itself infected) renders otitic infection quite a common thing.

Kerrison (Laryngoscope, Sept. '07) states that acute tympanic inflammation calls for adenectomy, for the following reasons:

1. The operation for adenectomy involves some risk to healthy ears. It seems wiser, therefore, to operate during the acute stage, when the ears can be safeguarded by a free incision of the drum membrane.

2. The abstraction of blood relieves tubal congestion and favors resolution.

3. With a growth sufficiently large to produce congestion of the pharynx, recovery from acute tympanic disease is apt to be slow.

4. Some cases will not recover until adenoid tissue is removed.

On account of the low level of the pharyngeal orifice, Preysing denies the easy exit of pus from the ear to the pharynx. He claims that the pus, which is mostly thick, "would rather, while

the infant is on its back, run into the mastoid antrum than through the tube."

Nearly all earaches in children under ten years of age, and the periodical earaches which occur in the night time, lasting for a few hours and recurring for a few hours for several days, are due to adenoid congestion. Indeed, this condition is the cause of febrile attacks more frequently than any disease of infancy save the exanthemata.

"Otitis media is of frequent occurrence in the very young. It may combine with the retrograde involution of the embryonal myxomatous tissue, which may disappear soon after birth, but often persists in the antrum and tympanic cavity and undergoes purulent softening." (Jacobi . . . Otitis Media in Children . . . Archives of Otology. Ap'l. '05.)

Microbes get into the middle ear in the contiguity of the surface of the mucous membrane quite frequently, or are thrown in during coughing, vomiting or sneezing (or especially blowing the nose); nurslings are in danger during suckling and deglutition. Medicinal and other injections into the noses are sometimes the cause of infection, more especially the snuffing up of solutions or powders.

In severe otitis media, scarlet fever leads (10 per cent.) Cerebro-spinal meningitis, the infection coming from within, produces hopeless deafness and staggering. Measles, diphtheria, influenza, pneumonia, enteritis, small pox are all diseases which may produce an otitis media.

Pain, dull to sharp, continuous or intermittent, sometimes with moaning, more often with a sharp cry, restlessness, pulling of the hair, burrowing of the head in the pillow, fever, sometimes stupor or convulsions are seen in the very young. Older children complain, the same as adults, of a feeling of fullness, tinnitus and deafness. There may be also tenderness of the ear on manipulation.

The principal thing, however, is the recognition of adenoids and their removal prior to infection, *and no age is too young.* In not a few instances, I have operated upon infants a month old. All babes that have difficulty in nursing and breathing, should be examined by the specialist, and if adenoids are enlarged, they should be immediately removed. Tonsils, at this age, should not be operated upon.

The symptomatology of adenoids in infancy differs considerably from that of childhood, but is still fairly characteristic. However, the evil results in infancy are even worse than they are in childhood.

There seems to be a prejudice in the profession, and among the specialists, of a certain extent, against the removal of adenoids in infancy, because of their likelihood of recurrence, but this should be of little weight, for if it be true, the infant is the gainer during the period of their absence. Their removal in infancy is accompanied by slight danger, and their removal in childhood is no more serious so that we cannot be justified in allowing infants afflicted with enlarged adenoids to have their development seriously or irreparably interfered with, when a simple operation, practically unattended by danger, will certainly to a great extent relieve them of these conditions.

The nose of the infant is relatively small and the respiratory space very small. The nasopharynx is low, but deep from in front backwards, being almost as deep as in the adult. The height increases rather rapidly for six months, and then slowly, up to two years. The nasopharynx is extremely vascular in infancy and the lymphoid ring is well developed.

Because of the low condition of the superior pharynx and post-nasal opening, a relatively small enlargement of the normal adenoid tissue may cause a marked obstruction to nasal respiration. In infancy, this is a very serious matter, especially when it is anywhere nearly complete. Interference with nasal respiration necessitates oral respiration, which the young infant performs very imperfectly, especially when asleep. This interference results in constant deprivation of a sufficient supply of oxygen, which in turn produces a disturbance of nutrition, which is uninfluenced by any method of feeding or mode of life.

Another serious result of nasal obstruction is the interference with suckling and sometimes with swallowing. The effort of suckling is so great that these babies take only enough food to satisfy the acute pangs of hunger, which again interferes with their nutrition and development. The difficulty of breathing makes them restless at night and interrupts their sleep, another disturbance to nutrition.

Snuffles in infancy is almost as diagnostic of enlarged adenoids as it is in childhood, and adenoids are almost always found in

those babies who suffer from frequent or continuous colds in the head. Irritating coughs, especially those occurring at night, without physical sign, are usually the result of adenoids.

Adenoids frequently accompany and probably cause catarrhal laryngitis and spasmodic croup. I say probably, because their removal so frequently relieves these symptoms.

An infant who is restless and sleepless at night, should, if definite cause for same be not evident, be examined for hypertrophied adenoids.

Adenoids frequently cause enlargement of the cervical lymph glands, and they are without doubt the greatest factor in the production of acute otitis media, consequently should be removed as a prophylactic measure.

PAPER:

THE INDICATIONS FOR THE MASTOID OPERATION.

HILL HASTINGS, M. D.

The subject of middle ear and mastoid suppuration has been discussed time and again in the meetings of ear, nose and throat societies. It is a subject of such vital importance that the chairman of this section and the program committee are to be commended for bringing it up for consideration at this general meeting of our state society. The part of the subject assigned to me—The Indication for the Mastoid Operation—is, I take it, to present for discussion the chief signs and symptoms that have been accepted by otologists in the last few years as indications for immediate operation.

In the beginning it must be admitted that some mastoid operations are done where recoveries would likely have resulted without operation. On the other hand it must, with equal fairness, be admitted that in some cases death resulted through unwarrantable

delay in operating. It should be stated, also, that seldom are the mastoid symptoms so severe that the patient would seek operation until serious if not fatal complications had arisen. It is not within the province of this paper to discuss these complications.

In every case of middle ear suppuration the mastoid antrum, being a part of the middle ear, is believed to be involved and there may be more or less inflammatory congestion of the adjacent mastoid cells. It is therefore fallacious to say, "when the mastoid antrum is involved the mastoid should be opened." This advice, I believe, is generally discredited both by the specialist and the general practitioner. No such arbitrary law can be laid down. In each case a careful study of many factors must be made to insure a timely operation. These factors may be determined by the following steps:

I. A careful otoscopic examination.

II. Examination of the mastoid region.

III. The history of the case and the general signs and symptoms.

I. *The otosocopic examination.* What can we learn from this?

First—The amount and character of the discharge, including a microscopic examination to determine the infective organisms.

Second—The appearance of the fundus, that is, the drum membrane and the adjacent canal wall.

Third—The degree of deafness.

A careful otoscopic examination is most important, for on that alone, without mastoid pain or tenderness and without general symptoms, such as fever, a mastoid operation is in a few cases indicated.

Permit me to cite one illustrative case. Mr. C., aged fifty-two, consulted on November 23, 1907, on account of profuse discharge from right ear of four weeks' duration. The onset was sudden; severe pain in the ear followed sore throat. The drum membrane perforated spontaneously and profuse discharge with relief from pain had continued; never any mastoid pain or soreness; no fever, nausea or vomiting; general health was good. Examination showed the canal full of pus; on wiping this away pus was seen welling out from a perforation in the drum membrane. A stained smear of the pus showed streptococci. The drum membrane was found to be beefy red, thick and sagging above and behind. The deafness was extreme (watch not heard on contact, whisper and

speech doubtful). No mastoid tenderness could be elicited, although the patient was warned of the importance of confessing any difference in sensation from pressure on the mastoid of each side. Operation showed that the interior of the mastoid was full of pus, the lower half of the process had broken down, forming one large abscess cavity that extended down to the knee of the lateral sinus. Smears from the mastoid pus also showed streptococci.

This case is not uncommon and is cited in order to illustrate the main diagnostic points to be looked for in an otoscopic examination, namely—profuse purulent discharge, the presence of streptococci, a badly inflamed drum membrane with sagging of the postero-superior wall and considerable deafness, all of which indicate a severe degree of middle ear involvement.

First. *As to the quantity of discharge.* When pus in large quantity continues to pour from the middle ear after three weeks of ineffective local treatment, the mastoid operation is indicated whether or not there are other signs and symptoms. On opening many mastoids one can not fail to be impressed with the fact that the destruction within the mastoid is, in many cases, surprisingly great, often having resulted in purulent collections on the brain or lateral sinus without any local or general signs or symptoms having been observed.

The *microscopic examination* of the ear discharge should be a routine procedure in every case. Smears should be made of fresh pus as it escapes from the perforation in the drum membrane. Of late it has been my practice to carry, ready for use, sterile swabs in glass tubes, from which cultures are made. This aids in the differentiation of the organisms. Smears alone are often unreliable. For instance, many reports of pneumococci are said to be inaccurate, culture showing the organisms to be capsulated streptococci. Furthermore, there are several and maybe many varieties of streptococci varying in virulence. Much of the past statistics of ear discharge infection is said to be valueless. It is an interesting and important part of the subject under discussion, but would be wearisome to treat of in detail. All observers seem to agree, however, that the presence of streptococci in the ear discharge indicates a dangerous infection.

Second. The *examination of the fundus*, comprising by that term the drum membrane and the adjacent canal wall, is of great

importance. A beefy red, bulging drum membrane with sagging of the postero-superior canal wall indicates a high degree of middle ear inflamation. This must be differentiated from the bulging, often attended by sagging, that is found in children with exudative catarrhal inflammation. In such cases the mucous character of the discharge, the pale redness of the drum membrane, the slighter degree of deafness, the absence of mastoid tenderness and the presence of less virulent bacteria serve to clear up the diagnosis.

Third. *As to the deafness.* In every case the hearing should be tested, at least by the watch, whisper and conversational voice. This is practicable except in very young children, and aids much when considered with the other signs and symptoms. The greater the degree of deafness, the more severe the middle ear inflammation, as a rule. So much for the value of the otoscopic examination. This is treated of first, because it is of the greatest importance, more important in my opinion than either the mastoid examination or the general examination.

II. *Examination* of the *mastoid region.* In the great majority of mastoid cases, more or less mastoid tenderness exists. In many cases of middle ear suppuration there is considerable tenderness during the first two or three days, especially when there is little or no drainage through the drum membrane. I do not believe that such tenderness indicates immediate mastoid operation. This tenderness is frequently due to intense vascular engorgement of the mastoid cells and may wholly subside in a day or two after good drainage is established through the drum membrane. It is the *persistence of mastoid tenderness* that is of serious concern, especially when it persists after the establishment of good drainage through the drum membrane. The tenderness is usually found by firm pressure at three points—over the antrum, over the tip and over the point of exit of the mastoid emissary vein. The tenderness is usually most acute during the first twenty-four hours, and decreases thereafter to increase again to great severity only when the outer table or its periosteum becomes affected. In such cases edema, of course, is found. Most patients lay great stress on the fact that their mastoid tenderness is not so severe as during the first twenty-four hours. This is not to be relied on as indicating that resolution is progressing without the formation of pus. Until all trace of tenderness is gone the patient is not safe, and even

then, the case is a doubtful one if the middle ear inflamation is not in like manner subsiding.

Tenderness over the mastoid is then not to be relied upon as the sole guide of the mastoid operation, but must be considered along with the otoscopic findings and the history of the case. The *time limit for the persistence of mastoid tenderness* can not be arbitrarily set. In no place does experience count for more in forming safe judgment. Delay, in order to be absolutely sure that pus in considerable quantity will be found, is manifestly unsafe. It is, after all, safety to our patients rather than a desire to prove ourselves infallible, that should influence our judgment. He who claims never to have opened a mastoid that would have recovered without operation, is a man of dangerous judgment. He has let many mastoids run too long, and in his record will appear some cases that resulted fatally because of unwarrantable postponement. What then is unwarrantable postponement? As previously stated, no arbitrary time limit can be set. Nevertheless, it is on this point that the general practitioner often wants some definite statement. We all meet cases where tenderness of several weeks' duration has existed, and yet abortive measures are still persisted in. While exceptions can be taken and are freely admitted, I believe it is good judgment not to delay over seven days from the onset of the middle ear abscess; provided, at the end of that time distinct mastoid tenderness exists, and provided there is no abatement of the middle ear inflammation, as evidenced by the persistent discharge and the otoscopic findings above enumerated. There are a few cases that will prove exceptions, some that will have to be opened earlier and some that can safely run longer and finally recover without operation.

III. The *history of the case* and the *general signs and symptoms.* In the history of the case, the following points are worthy of note,—mastoiditis following the acute infectious diseases is insidious in its onset and greater destruction is apt to result before pronounced signs and symptoms arise. This is likely due to the impaired resistance and is especially pronounced in scarlet fever, measles, and typhoid fever. A history of present or past tubercular trouble is important, for tubercular mastoid destruction is difficult to cure and recurrences are more frequent. A history of diabetes makes the prognosis more grave. The tendency is to delay operation in such cases, even though the mastoid indications are plain. When we

realize that in these very cases the resistance of the host of infection is poor it is all the more imperative to operate. The number of deaths from fatal mastoid complications secondary to scarlet fever, typhoid, diabetes, etc., prove the truth of this statement. Investigation of such cases will frequently show that there was unwarrantable delay in operating. I reported such a case at the last meeting of the State Society, wherein I was at least partially to blame, as a consultant, for the man's death. It was a case of diabetes in which middle ear abscess occurred. Operation was advised, but not sufficiently insisted upon. After six or eight weeks of apparent absence of mastoid signs, a sudden convulsion occurred and death followed in twelve hours. A large epidural abscess was discovered. There is hardly a doubt but that a timely operation would have saved his life. I reported a similar case at the American Medical Association meeting in July, 1905, with this difference, that the mastoid abscess complicated typhoid, and thrombosis of the lateral sinus was responsible for the death.

As to the general signs and symptoms, such as chills, fever, nausea, vomiting, etc., these are rarely met with in uncomplicated mastoid cases, except in children. In adults the general signs and symptoms are of slight consequence, until intra-cranial complications arise. In infants, however, high fever, vomiting and occasionally convulsions occur readily from middle ear suppuration. Such symptoms when otherwise not accounted for, should prompt a careful otoscopic examination. In adults, a slight but persistent rise of temperature occurring in the course of middle ear suppuration is suspicious of mastoid involvement.

As to a differential blood count, this is generally regarded to be of no value in determining upon the mastoid operation, although, of course, of great value in determining complications that produce general infection.

As to the use of vaccines—may we delay operation on the mastoid in order to await the result of curative vaccine injections, which have recently been advocated with some enthusiasm? I should like to have the opinion of those who have had greater experience in the use of vaccines in other suppurative processes. Personally while I am trying vaccines in suitable cases, so far I have seen no positive results. While the future may possibly hold much in store for us, I believe that at present it is dangerous to postpone a mastoid

operation when the indications are positive in order to await the result of vaccine injections.

In conclusion, a timely and thorough mastoid operation we should remember serves two distinct surgical purposes, namely, it cleans out the diseased mastoid, including the antrum, and in so doing, efficiently drains the tympanic cavity. The latter purpose should not be lost sight of in the discussion of this subject. No one who has opened or seen opened a hundred or more mastoids can fail to be impressed with the rapidity with which the tympanum regains a normal condition after the mastoid operation. Efficient drainage in the cure of infection of other cavities in the body has become a well established surgical procedure. In ear work it is of no less importance. It is not unlikely that we will see in the next decade fewer chronic discharging ears because of more prompt operating in the acute cases.

DISCUSSION.

Dr. E. W. Fleming: The time allotted for discussion is so short that it would be impossible to cover all the points brought up by Dr. Hastings in his most comprehensive and interesting paper. I am on the program to discuss his paper and therefore mention it particularly. I shall touch only briefly upon a few of the points dwelt upon by the essayists. If all cases of mastoiditis following acute otitis media coming to us for an opinion corresponded to that class in which all the classical inflammatory manifestations, both at the fundus of the ear and in the mastoid region, are present, the duty of the surgeon is plain. Recovery, however, even in this type of cases has occurred in my practice in patients who refused operative interference of any kind. Two types of mastoiditis frequently met with are, first, those cases where the manifestations of inflammation in the mastoid bone are especially pronounced—while the drum membrane and deeper canal appearances are at no time well defined—and second, cases where the very opposite conditions prevail. Another and quite the most difficult cases are those which show little or no adequate local or systemic manifestations to help us to determine the true condition of the patient. These cases where the indications for operative interference are not well defined must be studied from day to day in their minutest detail. Here the keen appreciation of

the personal judgment of the surgeon that comes from an adequate clinical experience is to my mind the most important factor in determining whether or not the case should be operated. A laboratory diagnosis may be very helpful. It should be made by one whose technique is free from error, else it may be worse than useless. It is exceedingly important to constantly keep in mind that intra-cranial complications of purulent otitis media may occur other than by way of the mastoid cells. As, for instance, the tegmen tympani—or it may be by way of the circulation. As good drainage is essential to the prompt cure of an acute otitis media, an early mastoid operation, even in doubtful cases, has the advantage of checking further destruction by the disease of the middle ear structures by establishing posterior drainage, and at the same time permits the surgeon to make sure of his ground from the view point of the mastoid cavity. Having had several disagreeable experiences in cases of brain, meningeal and sinus involvement following acute suppuration of the middle ear with associated mastoiditis, without mastoid symptoms, I am strongly disposed to agree with Whiting, Jackson and others, who contend for early, and, it may be, unnecessary operation, rather than assume the responsibility of a possible development of a dangerous complication as the result of an over conservative attitude.

Dr. Edw. Gray, Eldridge: The first case of mastoid disease which I ever saw was in the year 1875. At that time you are aware the state of medical education was quite different from what it now is, and it was because of the occurrence and the presenting before me of a case of fatal mastoid disease in the person of a girl 16 years of age, taken into the hospital, and the evident helplessness of the hospital force in fighting it that led me to go to the city of Vienna and there study diseases of the ear, nose and throat. I will not worry you with the history of that case, suffice it to say that it was a case of neglected ear disease following measles in childhood. The otorrhea had been running on for a period of perhaps ten years, and I at the time was Senior Assistant in the Presbyterian Hospital in the city of New York, and I was sent down to the tenement district to examine this case to see if it were a proper one to enter the hospital, because evidently the suspicion of the authorities was that it might be a case of cerebro-spinal meningitis. The symptoms were cerebral. The girl was

taken into the hospital and after a variable history over something like ten days finally died with symptoms that resembled apoplexy more than anything else. A post mortem was secured. The tegmen tympani was thoroughly disorganized, blackened, and the orifice through which the pus, etc, had made its way was clearly defined, and the portion of the cerebral meninges which had thereby been irritated and formed an abscess of the brain was clearly demonstrated.

After returning from Vienna and coming back to this, my native state and entering into practice it was a long time before I encountered another case. I refer to this because it was a singular case. There has not been a single reference in all that has been said this afternoon to the conditions that case presented. It was evidently a case of acute purulent otitis media with mastoid complication. At the time I was called it was only because the patient had been under the care of another practitioner who was, by the way, a medical officer in the United States Army, and I am sorry to say he was one of the very infrequent class in the army who sometimes went off on a spree, and because he was incapacitated I was called. I found the patient was treated simply for neuralgia for something like three weeks, and yet it was a case of acute mastoiditis which any one who knew the symptoms could recognize. I immediately explained to the family the necessity of operation, and how much more difficult that was in those days. Of course they wanted consultation. The consultant was friendly and told them they were losing valuable time. He told me, "I don't know anything about it. If you do, go ahead." I went ahead and removed some fifty minims or more of pus and the patient recovered with no untoward symptoms so far as hearing was concerned, but with the complication of an abscess burrowing down the sterno-mastoid. The patient finally recovered and is alive to-day. He has said to me that that was the worst thing I ever did for him, for he ought to have been allowed to die, and the community thought so too.

CHAIRMAN ROBERTS: Where did you open, Doctor, the mastoid?

DR. GRAY: Yes sir, the mastoid, with a drill.

DR. F. L. ROGERS, Long Beach: I wish to refer briefly to a phase of the etiology of acute inflammation of the middle ear, which has

been touched upon by two or three of the papers read: i. e. that adenoid growths are a frequent, perhaps the most frequent cause either directly or indirectly, of acute otitis media in children, and to emphasize what has been said relative to their early removal.

When I was a student the teaching was, that the operation should be postponed until the child had reached seven or eight years of age in order to give nature a chance to "absorb the growths." But when I had a little experience of my own, I found as others have, that once the third tonsil is hypertrophied in a vast majority of cases, it remains so, and the sooner the obstruction is removed the better for the child. And the age of the child cuts little or no figure. These children are nearly all anaemic and badly nourished, and if they live to reach school age are found markedly below par in mental and physical development. They are mouth breathers, and from disuse the nasal passages and dental arch fail to expand normally. This leaves the child more or less permanently crippled and deformed and unless the post-nasal obstructions are removed early in the developmental period of his life these deformities remain a permanent handicap.

The adenoid operation, if properly performed is a bloody and shocking procedure, and in my opinion is rarely done with thoroughness unless under a general anesthetic. I regard the operation without general anesthesia both dangerous and barbarous, and rarely thoroughly executed when undertaken in that way. To do the operation well considerable skill, and great care is necessary, and a digital examination of the field of operation should always be made after the curettement. Particularly would I call attention to the difficulty of reaching Rosenmueller's Fossae and the field around about the orifices of the Eustachian tubes and the lateral walls of the pharynx, with the Gottstein curette.

For this supplementary work I have found a good instrument in the curette devised by Byington, of Battle Creek, and made by F. A. Hardy & Co., with a long slender loop blade and a double cutting edge, placed horizontally. I never consider an operation complete until I use my long straight handled curette through the nose, my finger at the same time in the naso-pharynx as a guide to remove any small excrescences on the lateral walls of the posterior nares, or small vegetations on the turbinates. In fifteen years I do not remember to have had to operate the second

time on a case when these precautions were all taken and a general anesthetic was used.

Dr. Fred Baker, San Diego: I want to emphasize one or two points. We have had the question of the radical operation for adenoids mentioned by Dr. Rogers and I fully concur in what he said, but he speaks of the curettes and their inefficiency. I think it has been the experience of every one who operates that the use of the curette is unsatisfactory if the operation terminates at that point. I thoroughly believe the best instrument for operation on adenoids is the finger nail. Of course it must be rendered as aseptic as possible, but I do not believe any operation is satisfactorily done unless you examine with the finger to see what is done, and I do not believe the Gottstein curette or anything else will clean out the cavity so that the adenoid will not return. I am of the opinion—I do not think my experience is wide enough to state positively—that in the removal of adenoids a second operation is necessary only where adenoids are left after operation. I believe if you go in with the finger nail and clean out the cavity as well as you can you will have few cases of recurrence of the adenoids.

In this connection I wish to call attention to a method of procedure I have never seen mentioned or noted before. It is original as far as my own experience is concerned. On one occasion in cleaning the finger nail I pared down the inner surface until I had produced a chisel edge, and I was surprised at the facility with which I could remove the tissue. I am tempted to try the complete operation in this way.

I want to protest against the removal of the tonsils and adenoids in the same operation. I have seen it done a number of times and I have done it myself twice. In both of these instances, owing to the fact that the hemorrhage was considerable, and it was impossible to continue the anesthetic, I did an imperfect operation. I believe the removal of the adenoids is the most important and the tonsils can be removed afterward. The operation should be divided and the tonsils removed either before or afterward. Take plenty of time and use general anesthesia every time, if it is possible, and you will generally prevent recurrence.

Dr. F. D. Bullard, Los Angeles: From personal experience the best way is to pass the Eustachian catheter. Having twice

had otitis media, my colleague, Dr. Ellis, inflated the ear and all the symptoms disappeared, and I urge your attention to early inflation.

A Member: Do you apply that to children?

Dr. F. D. Bullard: I don't know that you could apply the catheter, but Politzerization would be possible. Another thing is to urge upon the general practitioners, to send their children to the specialist to have their adenoids taken care of. Not long since a physician in Los Angeles sent his child to me for an error in refraction, and I called his attention to the fact that the child had adenoids. He had not recognized it, although he is a competent physician. Now I believe we should go around and have the doctors all in and lined up first.

Dr. William Simpson, San Jose: I would like to say in regard to the use of the Politzer bag that I have been using it for twenty-five years and I have never seen any trouble from it. In many cases I think it is the very first thing to do. But there is just one point I would like to make in that connection, and that is that we are making a tremendous mistake in discussing these questions among ourselves as specialists; that to do good this discussion ought to take place when the general practitioner is present, for I am sure there is not a man in this room but that knows that a campaign of education ought to be conducted on this line for the benefit of the general practitioner. The difficulty comes from the fact that they hang on to these cases just as long as possible, and send them to us so late that it is almost impossible for us to do a bit of good. I heard a lot here this morning as to a campaign of education on the milk question, and on various other questions, and if there is any question on which a campaign of education should be conducted, it is "that the general practitioner does not know everything about eyes and ears." But until we get to that point you might just as well take your chance, and when they come to you do the best you can.

Chairman Roberts: I will say in reply that this is supposed to be a meeting for the general practitioner and not for the specialists.

Dr. William Simpson: I am glad if some of them heard what I have said.

Dr. R. L. Doig, San Diego: Doctor, the remarks you have just made make unnecessary much of what I wished to say.

Watching the work of specialists for the past ten years, the results have been so impressive, so astonishingly beneficial in many cases, that I am anxious the general practitioner should be educated as to its importance.

During the first fifteen years of my practice I was practically ignorant of this work and its results. Two years ago I presented a paper at Riverside from the standpoint of the general practitioner. Very much to my disappointment I read it before a lot of specialists who knew more about it than I. That was not what I wanted to do, and the effect of the paper was lost. Another thing I would urge, and that is the directing of the attention of the dentists to this matter. In that paper I said that one of the best papers I had heard was a dental paper, in my town, on this subject, but I have reason to feel that dentists in general do not appreciate it. Just the other day a little girl, probably fourteen years of age, moved into a house adjoining mine. She is one of those rather pretty girls, spoiled by prominent front teeth She has a pretty voice and would sing well if she had proper respiration. I called the attention of her mother to these prominent teeth and she said, "Yes, it is a case where she sucked her thumbs, and the dentist said, as long as she did, he could not do anything for it." I called her attention to the fact that it was because she could not breath through her nose. She said she had never heard of adenoids and the dentist assured her that the trouble all came from the child sucking her thumb.

Dr. F. L. Rogers, Long Beach: In as much as this is a general practitioners', as well as a specialists' meeting, I fell that the remarks of Dr. Simpson should be commented upon, particularly in reference to the use of the Politzer bag in acute otitis media I do not believe this section wants to go on record as favoring indiscriminate, or even routine Politzerization for this condition. The doctor stated, whether as a matter of inadvertence or not, that the first thing that should be done in a case of acute otitis media is the use of the Politzer bag.

Dr. William Simpson: I did not say in every case; I said often it was the first thing to be done.

Dr. F. L. Rogers: I cannot agree with the statement even with that qualification, for in my opinion the first thing indicated in acute otitis media is to cleanse, and as far as possible to sterilize the naso-pharynx as well as the external auditory canal, with hot saline antiseptic solutions. This in itself depletes the tissues and relieves the tension, and may be sufficient to permit the entrance of air by natural means. If this fails then use a Politzer bag early, but with extreme caution. I thoroughly agree with the gentleman who said, that the use of the eustachian catheter even in children, is the most satisfactory way to inflate the middle ear. Only yesterday I used it for a child four and a half years old. A small amount of one per cent cocaine solution was used first and the child made but little complaint, and the results were very satisfactory, both as to pain relief and improvement of hearing.

Dr. Cullen F. Welty: I believe that all cases of acute purulent otitis media not dependent upon infectious diseases should have their tonsils and adenoids, particularly their adenoids, removed at once, as they are the predisposing factor in so many cases, and if not removed the ear may continue to discharge indefinitely, or the hearing be more or less seriously impaired. No doubt all of you have seen such cases. If there ever was an indication for removal of the tonsils and adenoids, this should be considered the one of greatest importance. Over treatment of the naso-pharynx in acute infectious diseases I believe is responsible for many cases of purulent otitis media. I should not recommend more than two or three treatments per day, and of the mildest form that can be applied. I sometimes doubt the advisability of any interference. When the tonsils and throat are seriously infected it may do some good.

Politzerization in acute otitis media is the thing par excellence

Politzerization in acute purulent otitis media does not do any good, nor does it do any harm. Many ear men have contended that the pus was blown into the mastoid cells. If you only recall pathology you will remember that, the two, the tympanic cavity and the mastoid cells, are infected primarily in this disease.

Some recommend Politzerization two or three days following a free incision of the drum membrane, in order to forcibly blow some of the pus from the tube and tympanic cavity. In this condition it is also stated that a mastoiditis may be produced, which is entirely wrong as I have stated before.

I do not believe Politzerization does much if any good at all because the tube enters about the center of the cavity, and that pus below the center will remain.

The question of a bandage in acute otitis media has not been spoken of, I wish to recommend it, and insist that you are not doing your best when you allow your patients to go without it. As a physician or surgeon, would you allow your patient to go about with an inflamed part without protecting the same? And I say a piece of cotton put into the ear is not sufficient protection.

The only criticism I have to make on Dr. Hastings' paper is that it is not sufficiently complete.

The question of heat and cold in acute purulent otitis media has been discussed for the last ten years by ear surgeons, and when I say that most of them have discarded the cold in preference to the heat you must accept it as such.

I remember very well a case of acute otitis media that terminated fatally, was treated by the coil with ice water. This made such an impression on me that I will never forget it. The symptoms were so masked by the use of the coil that another case was sacrificed to science.

I believe in hot applications because they have served me well.

As I have said in my paper, after a free incision of the drum membrane, put on a hot solution, (preferably Burrow's, because it is an astringent antiseptic), and if your patient is not improved in twenty-four hours the most conservative procedure is that of operation.

In connection with the use of heat, Dr. Thomas says I will increase my cases for operation. This has not been so, as I have had but two cases of acute purulent otitis media that I have seen from the acute condition to increase in severity and demand operation. I most heartily recommend hot applications, and will say, if you use it in ten cases as I have advised, I am confident you will never use cold again.

Dr. Pischel takes exception to my statement, that "a competent man can only do good in mastoid surgery." I will answer to this by saying, that I have never seen the facial nerve cut. I have seen the dura uncovered and punctured if you please, the sinus uncovered and accidently opened, but I have never seen a serious complication nor a complication of any kind, as the direct result of the operation.

I have seen acute cases of mastoiditis die because they have not been operated upon early enough, but from no other reason.

The deaths that come from anesthesia have nothing to do with the operation at all. That is entirely foreign to the subject.

In a two years' service in the Politzer Clinic, there were about forty-five deaths. The lesions were always found to be in the brain or its membranes and from delayed operation in either the acute or the chronic condition.

One gentleman spoke of a fatal case on the third day from cerebral complications. This again illustrates the very points that I have tried to emphasize. I am confident had this case been examined carefully, and conditions looked for that I have spoken of, the case would not have been sacrificed.

Dr. Kaspar Pischel, San Francisco: It has been pointed out before that the family physician should examine or have examined a child's ear in every case of fever where he cannot find any other explanation. In this connection I wish to call your attention again to a symptom which Sanford Blum pointed out last year. It has been mentioned in some of the text books but has not been brought to the attention of the family physician. It is the sensitiveness to pressure behind the jaw. When examining a baby you can easily try it.

While I perfectly agree that we should make microscopic examinations of the discharge from the ear, I do not think we are far enough advanced in bacteriology to judge about the seriousness of the case from the organism found. We find frequently that staphylococcus infection leads to serious troubles, while a pure streptococcus infection may after paracentesis pass off in a day.

As to the closing sentence of the last essayist, *"and I am confident they will not if operated on early enough."* I take exception to this statement. I think there are acute infections which spread so quickly that we cannot possibly make the diagnosis early enough. *"In mastoid work a competent person can only do good or* ———," as he said at the reading of the same paper in San Francisco the other day, "can never do harm." I have to take exception to this too. Even the most competent surgeon can do harm. In mastoid work we have to resort to general anesthesia, and there lies the chief danger. The last few years a number of cases have been reported

in which death occurred several days after general anesthesia from auto intoxication. That can happen in mastoid as well as in abdominal work, and I am sorry to say that I have had one such experience myself.

MEMBER: I should like to ask whether with chloroform or with ether?

DR. PISCHEL: According to a paper read by Dr. Bevan of Chicago, at the Portland meeting, chloroform is the more dangerous, but even ether can cause acute affections of the liver and kidney several days afterward.

MEMBER: I am asking whether your experience was with chloroform or with ether?

DR. PISCHEL: In my case chloroform had been used. The operation lasted not more than three quarters of an hour.

PAPER:

THE STATUS LYMPHATICUS, WITH PARTICULAR REFERENCE TO ANESTHESIA IN TONSIL AND ADENOID OPERATIONS.

W. HOLMES ROBERTS, M. D.

In and about Los Angeles, so many deaths have recently occurred under general anesthesia, either during or immediately following minor operations, such as the removal of tonsils and adenoids, that it seems advisable to look into the subject to see if such sad occurrences can be prevented in the future. The subject of deaths from anesthetics has been thoroughly gone into in the past, and, by most investigators, it has been ascribed to a condition existing in the patient which has been termed the "status lymphaticus" or the "lymphatic constitution." I make no claims for originality for what I am about to present, but I simply wish to set forth some well established facts in connection with the status lymphaticus, with the hope of thus refreshing the minds of all present, in order that we may be placed more thoroughly on our guard, and

that we may thus prevent deaths which are bound to occur if anesthetics are to be used as indiscriminately in the future as they have been in the past. Few physicians are capable of giving an anesthetic, and no one without medical training should *ever* give one. Nothing is more erroneous than the idea that anyone can give an anesthetic. Doubtless many deaths under anesthetics have been caused by gross or almost criminal carelessness.

This paper is to deal solely with those cases in which death has occurred when the anesthetic has been carefully administered by a proper anesthetist, and in which, in all probability, the status lymphaticus existed, as is shown in the report of one autopsy. In nearly all cases in which the status lymphaticus has been demonstrated at autopsy, enlargement of the thymus gland has been found. This enlargement, associated with sudden death, was first mentioned by Bichat in 1723, but the connection was then apparently overlooked until 1829, when Copp called attention to it. Little notice was paid to it until 1888, when Jacobi, in this country, and Grawitz, in Germany, took it up.

In 1889, Paltauf, of the Vienna School, wrote: "Hyperplasia of the thymus is physiologically, as well as anatomically, an element of a general hyperplasia, and is a result of a derangement of nutrition or metabolism which also causes a degeneration of the cardiac centers." He found at autopsy, in addition to enlargement of the thymus, a hyperplasia of the entire lymphatic apparatus; enlarged nodes all over the body; of faucial and lingual tonsils; of the intestinal follicles; enlargement of the spleen and hypertrophy of its follicles; changes in the circulatory system. Here he noted a true hypoplasia; the aorta and small arteries were smaller and thinner than normal, and there were signs of cardiac dilatation.

Kolisko,[1] who averages some 2,000 autopsies each year, with about six of these dying from cardiac paralysis due to chloroform, says: "In these cases we always find the condition known as the 'habitus lymphaticus.' The nature of this condition is (1) a persistent thymus gland, which has often become considerably enlarged through an increase of its lymphatic tissue; (2) enlarged lymph glands; (3) adenoid vegetations in the pharynx, enlarged follicles at the root of the tongue and in the pharynx; (4) enlarged follicles in the intestines and stomach. These conditions are accompanied by some dilatation of the heart; with no changes in the muscles or endocardium; or occasionally there is evidence of a pre-

vious cardiac dilatation, marked by thickening in the endocardium, but not recognized clinically. There is also very frequently found a hypoplastic condition of the arterial system."

Is it possible to recognize the status lymphaticus prior to the beginning of symptoms which generally terminate in death? Many physicians, especially those of the Vienna School, claim that it is in some cases. Blumer[2], quoting from Escderick, who is one of the foremost authorities, says: "The subjects of the status lymphaticus usually have a pale, thin skin, a pasty complexion, and a good pad of subcutaneous fat. Frequently signs of rachitis or scrofula are present. The superficial lymph glands, especially those of the neck and axilla, are enlarged. There is hypertrophy of the tonsils, the circumvallate papillæ of the tongue, and the pharyngeal lymphatic apparatus (adenoids). The spleen is often palpable." There may be faulty development of the sexual organs, a thymus capable of percussion, and an increase of the lymphocytes in the blood. We should be very suspicious if, in addition to the foregoing, we obtain in children a history of dyspnœa and laryngismus stridulus, and in adults a history of fainting attacks. It should also be kept in mind that rachitis and goitre are often associated with the status lymphaticus. It would seem reasonable to suppose in this connection that the status lymphaticus is probably the cause of the frequent deaths occurring after operations for goitre. It is thus seen that hypertrophy of the faucial and pharyngeal tonsils are among the symptoms found in the status lymphaticus. We must remove these enlarged glands, when of sufficient size to interfere with the growth and development of the patient, with the least possible risk and shock.

Blumer[2] says: "Individuals who are the subjects of the status lymphaticus are born with an instability of the mechanism regulating the so-called 'horror autoxicus,' at any rate, so far as the lymphatic apparatus is concerned, so that they are subject to intermittent attacks of lymphotoxemia which may lead to reflex nervous phenomena of various kinds, or may cause death from cardiac paralysis. During the attacks of lymphotoxemia, such individuals are especially susceptible to the action of the bacterial or chemical poisons, and also to physical and psychical shocks, which at these times may cause their death under circumstances which would be trivial to a normal individual." This would explain the deaths which have occurred following the injection of remedial agents as

in the well known case of Prof. Langerhans' son, who died soon after receiving diphtheria antitoxin hypodermically. This would also explain deaths following shocks, frights, trivial injuries, or after diving in cold water.

Blumer,[2] in concluding his comprehensive paper on the status lymphaticus, says: "(1) The condition known as the status lymphaticus is a definite pathological entity. (2) It is probably associated with, if not due to, a condition of intermittent lymphotoxemia. (3) It may be associated with sudden death; probably, as a result of lymphotoxemia alone in some cases, or as a result of the action of toxic, physical, or psychic injuries, which are rendered much more powerful than usual by the predisposing action of the lymphotoxemia. (4) In some cases the sudden death is undoubtedly mechanical and due to asphyxia from pressure of the enlarged thymus on the trachea."

Olmacher[3] says that many deaths from status lymphaticus are due to increase of intra-cranial pressure with sudden edema, the clinical picture depending on the site of the edema, and the resulting pressure. If the external surface of the cerebrum is involved, various convulsions of the motor apparatus occur; if the floor of the fourth ventricle, sudden cardiac or respiratory failure.

Most surgeons admit that to remove tonsils and adenoids properly, general anesthesia is necessary. What anesthetic is safest? Halstead,[4] in an able paper read before this society at its general meeting in Philadelphia, 1900, showed clearly the necessity of performing the adenoid operation on children under some sort of general anesthetic, in order to avoid the great shock to the child's nervous system, which would occur where no anesthetic is used, or when only cocaine is used locally. In addition to the fact that this operation cannot be thoroughly performed unless the child is unconscious, the shock would be sufficient to cause the death of any patient with the status lymphaticus, and this should condemn it. Halstead points out the great danger of giving chloroform to any child with adenoids or enlarged tonsils, whether being operated for them or for something else. The lymphatic tendency is most marked in children before puberty, and it is during this time that chloroform is particularly unsafe. The saying that chloroform is the best anesthetic for children is amply refuted by statistics. Chloroform is a decided depressant, and when with children there is added fright with violent struggling against the anesthetic, we

have a combination that can readily bring on cardiac syncope in a patient often predisposed to it by the status lymphaticus. This complex is undoubtedly the cause of so many deaths under chloroform before operation is begun.

Sanford[5] has reported a death following the removal of adenoids under cocaine. Were cocaine more generally used, many deaths would undoubtedly occur, as young children are quite susceptible to its toxic effects.

Dr. W. J. McCardie,[6] in a recent article, reports thirty deaths from anesthesia, in which the status lymphaticus was demonstrated. These occurred as follows:

Following chloroform	17
Following ether	6
Following chloroform and ether	5
Following nitrous oxide	2
Following ethyl chloride	0
	30

Thus, in twenty-two out of the thirty, chloroform was used. The youngest patient was six months, the oldest fifty-five years. Twenty-four were under twenty years.

Dr. John Wyeth,[7] of New York, performs seventy-five per cent of his operations under chloroform, but when operating on children he uses ether, as he considers chloroform too dangerous.

McGuire[8] says: "Recent investigation, however, seems to show that ether is equally as dangerous (as chloroform) under similar circumstances." With this I cannot agree.

Dr. Hill Hastings, when house surgeon at the New York Eye and Ear Infirmary, went over the records of tonsil and adenoid operations, covering thousands of operations which had been performed during a period of five years, and he did not find a death. Indeed, I believe they have never had a death from the anesthetic in this operation, and ether is invariably used.

In personal communications from Dr. E. A. Crockett and from Dr. Philip Hammond of Boston, they report that they have never known of a death in Boston from ether in the tonsil or adenoid operation. I may add that, in Boston, ether is always the anesthetic used, and that the patient is placed in the upright position for the operation.

Among the thousands of operations covered by the reports of Hastings, Crockett and Hammond, there must have been some operations on children with the status lymphaticus; and yet, among these reports, there is not a fatal result, and all were operated upon under ether.

A few miles from Los Angeles, in a seaside town of some 10,000 inhabitants, there have been three deaths within a year—these occurred during tonsil or adenoid operations, and chloroform was used in each case.

In and about Los Angeles, the following deaths have occurred as the result of anesthesia. In only one case was autopsy permitted, and in this the status lymphaticus was found. In several others there is no doubt that this condition was present, but in absence of autopsies no positive claims can be made.

I wish to thank Dr. F. D. Bullard of Los Angeles, for his kindness in sending me the reports of most of these deaths.

CHLOROFORM.

Case 1. Boy, aged thirteen. Operation—Removal of tonsils and middle turbinates. The anesthetic was taken nicely, and the operation was completed with the patient in good condition. His pulse then became irregular, breathing shallow, and face suddenly blanched; after the beginning of artificial respiration, he gasped two or three times, and then expired.

Case 2. Child, aged three. Operation—Removal of adenoids. The surgeon had just started to remove the adenoids when it was noticed that breathing had stopped. It was then found that the heart had ceased beating. This child had previously had chloroform administered for circumcision. Was this a case of intermittent lymphotoxemia?

Case 3. Child. Operation—Removal of adenoids. The surgeon would-not reply to my letters asking for details of the case.

Case 4. Child. Operation—Removal of tonsils and adenoids. This case is reported in the words of the surgeon in whose hospital service it occurred. "The patient, a girl twelve years old, was brought into the operating room at the ——— Hospital, anesthetized and ready for me to operate. I had never seen the patient before. My memory is that she was a healthy looking girl of about twelve years of age, of medium build, neither fat nor thin. The patient was not fully under the anesthetic, so operation was de-

layed until the anesthetizer stated she was fully anesthetized. My recollection is that not a long time elapsed before operating and not much anesthetic given, but I was not paying attention to that part of it. I remember, however, that there was no struggling during the removal of the tonsils and adenoids. The operation itself took a very short time, I should say not over three minutes. On completion of the operation I was struck at once by the extreme pallor of the patient and the fact that she was not breathing. There was no cyanosis. I do not remember whether the pulse was beating or not, nor whether the pupil was dilated or not, or responsive to light. Artificial respiration and other measures were promptly used, but without the slightest sign of return of the respiration or the heart beat. No autopsy was secured. Chloroform was used because I had failed to tell the interne my preference for ether. The interne told me he had examined the chest and that the heart was normal. The patient had only been in the hospital an hour or two, so that nothing was known of her."

Case 5. Adult. Operation—Extraction of tooth.

Case 6. Woman. Operation—Dressing surgical wound. Ether had been used at the operation.

Case 7. Adult. Operation—Lancing of carbuncle on the neck.

Case 8. Adult. Operation—Avulsion of the toe nail.

Case 9. Adult. Died while preparing the abdomen for operation.

Case 10. Adult. Died before beginning the operation; fear probably figured largely in this case.

Case 11. Adult. Tonic spasm of the jaw, and probably "swallowing of the tongue." The attendants were unable to open the mouth.

Case 12. Adult. Lancing of carbuncle.

Case 13. Adult. Uterine curettement at early stage.

Case 14. Adult. Uterine curettement at the end of the operation.

Case 15. Adult. Hemorrhoids. Death occurred during dilatation of the sphincter.

ETHER.

Case 16 Woman, aged thirty-six. Death occurred from edema of the lungs, one hour after a prolonged operation.

BROMIDE OF ETHYL.

Case 17. Woman, aged twenty-three, native of Sweden. Operation—Extraction of teeth. Three minutes after administering two drachms of bromide of ethyl, extraction was begun. One tooth had been removed with some difficulty, when the patient blanched and breathing became shallow. Artificial respiration was commenced, giving slight improvement of color, which immediately became bad again. Dr. Stanley P. Black made an autopsy, and his report is as follows:

"Body well nourished; subcutaneous fat tissue well developed. On removing the sternum, the thymus gland was situated beneath the manubrium, size 3 by 2 1-2 by 3-8 inches. Lungs normal. Heart—left ventricle well contracted, right ventricle flabby; cavity empty, valves normal. Liver normal. Spleen somewhat enlarged. Pancreas normal. Kidneys normal. Intestinal tract, the solitary follicles at base of the tongue in esophagus, stomach, duodenum, jejunum, ileum and colon quite prominent, standing up under mucosa like shot. Peyer's patches likewise swollen. Glottis normal. Diagnosis—Paralysis of right ventricle. Status lymphaticus."

The records of the deaths of some of the patients under chloroform anesthesia have shown a history of having successfully taken this anesthetic once, only to succumb to a later administration of it. An intermittent lymphotoxemia could easily account for this, the lymphotoxemia being in abeyance at the first and present at the fatal administration. Whether or not death would have occurred in these cases if ether had been used, it is impossible to say. We do know, however, that in most of the fatal cases, cardiac paralysis occurred: and that chloroform is a cardiac depressant, while ether is a cardiac stimulant.

The status lymphaticus is most common in the first decade. Is it merely a coincidence, then, that in this decade a greater proportion of deaths occur from chloroform than from ether?

SUMMARY.

1. We should always keep in mind the possibility of the status lymphaticus being present in children who have enlarged superficial lymph glands, adenoids, or signs of rachitis.

2. In the status lymphaticus, *all* anesthetics are dangerous, but particularly, chloroform.

3. In *all* operations on children, avoid *chloroform.*

4. In operations for the removal of tonsils and adenoids, ether is the safest anesthetic to use.

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DISCUSSION.

Dr. F. D. Bullard: Mr. President, and Gentlemen of the Society—Although I reported these cases to Dr. Roberts I am not responsible for them all. One, however, was my case. This patient died either from the anesthetic or from some other cause. I think it was shock. An autopsy was held. The patient blanched. She was not cyanosed. Artificial respiration accomplished nothing, which is characteristic of that form of death. Any anesthetic to be given in status lymphaticus should be a heart stimulant. Bromide of ethyl is a heart stimulant. Then, as a rule, it is safe. Under ether the patient becomes red in the first stages at least, and in giving anesthetics in adenoid operations if you use Wyeth's hot air method of giving ether, heating it, you will only have a prolonged first stage, and the operation can be done then, and I believe there is no record of a death under primary stages of ether anesthesia. Chloroform under no circumstances should be given in any operations about the face that occasion a great deal of pain, as extraction of teeth and the removal of adenoids do. I have now given anesthetics in nearly 700 cases, and I become more and more an ether man, and do not give chloroform to children. I agree entirely with Dr. Roberts. I may give a very few whiffs of chloroform, perhaps preceded by cologne, to sort of get the patient going, but I go right on with ether, and I can get quicker results with ether than with chloroform. Then again the after results of ether are not so bad as with chloroform very frequently. You do not have that fatty degeneration of the liver which will come from chloroform more frequently than from ether. I believe with the

doctor that only a physician should give anesthetics, and I believe also a person who has had a great deal of experience, for there are five things to be watched continuously in all anesthetics, namely, the respiration, the pulse, the color, the condition of the pupil and the muscular relaxation. If you have these five things in perfect accord you are safe. If one is not good, look out. If two or three of them are bad you are in danger. If any one of the majors is bad, look out. If with dilatation of the pupil you still have mobility of the pupil and a redness of the skin and increased heart-beat, you are in a safe condition. But on the slightest sign of blanching the anesthetic should be removed. Once in about a hundred cases bromide of ethyl causes blanching. If they don't turn red I throw away that anesthetic. Chloroform frequently may be a heart stimulant, but it so frequently causes blanching that I have concluded to throw it out. But for quick operation never give chloroform. It is dangerous, because a large percentage of the deaths come early. Only one of these 20 deaths around Los Angeles occurred late in the operation, and that person had been on strychnia for some time and had come to the hospital and quit the strychnia and the physicians didn't know that, and therefore was in a condition under par. That person had three times before taken chloroform. It was an adult and might have been an intermittent intoxication, as Dr. Roberts describes.

Dr. S. P. Black, Pasadena: The subject of the status lymphaticus is of extreme importance to the operating physician, and to the physician after death of a patient. In the first place it is of the utmost importance to be able to recognize the condition. That, however, is sometimes difficult. Oftentimes we find cases in persons of robust constitution apparently. One of these cases I made an examination of. He was a strong man, sexually almost over rather than under par. He was walking along in the dark and struck his hand against a table. In two minutes he was practically dead. He was picked up dying. In that case the coroner gave a certificate of heart disease, but the subject went to autopsy, which I made, and it showed a typical case of status lymphaticus. He was about 36 years of age, but I got a history afterwards that he had been subject to fainting fits from his youth. One time he struck his knee against a chair and fell over and was unconscious for an hour. In that instance if he had had an anesthetic during one of those attacks he would have died. In these cases the surgeon

is often blamed for it. In Vienna I had a conversation with Kolisko. He said that all cases dying under an anesthetic had an autopsy in the medico-legal department. If the status lymphaticus was present it was counted an accidental death and the physician was not responsible. If we can find some way to recognize this condition it will be of very great importance. We know that it more frequently occurs in children, because most of these deaths from status lymphaticus are in the adolescent. Then in children, especially with adenoids, and especially if the spleen is enlarged or the superficial glands are enlarged, we should be extremely careful of any anesthetic, and with the history of anesthetics we should use only ether. Personally I am always afraid of chloroform. And the fact that even a young patient with this condition has had chloroform does not mean that he won't die the next time. I remember, when in the hospital during my interne service, a boy had been given chloroform repeatedly in a hip joint dressing which was extremely painful. It was given by the junior interne in any way. He seemed to be immune. But one time he was dressed and the house surgeon was giving the anesthetic, using extreme care. After three whiffs the heart stopped and the boy was dead. In those days we knew nothing about the status lymphaticus, but this was probably such a case. That shows that previous anesthetization does not show that the next time the patient will not die. So that we should not use that argument in favor of chloroform. The responsibility of the physician is certainly very great in these conditions and we should avoid cardiac depressants of every kind. Now it is not only from anesthetics, but from trivial injuries such as would not produce death in an ordinary adult, as in the case I spoke of where he struck his hand. Many cases are reported by Poltauf where persons had fallen into the water but didn't drown—the lungs were free from water—but died of cardiac paralysis due to the condition of status lymphaticus.

Dr. F. W. Miller, Los Angeles: I believe this paper is of as much importance or more, and more timely, than the ones that preceded it. While there is not much that is new in the others this is comparatively new and something we have overlooked entirely in our operative work. I personally would like to thank Dr. Roberts for the paper, because it brings up a subject worth while.

I want to speak of one thing, and that is the necessity for an anesthetic in nose and throat work, particularly in children. The doctor that preceded me said that he is taking out adenoids under local anesthesia. It is not so much because of the pain or discomfort as of the sight of blood and the nervous shock the child experiences, that I insist upon general anesthesia in children, and have, since the status lymphaticus has been brought to our notice, used ether exclusively. I will say, however, that I have used chloroform in probably several hundred cases with no bad results, but since the status lymphaticus has come before us I have stopped it. I have found, also, the position of the patient, either prone or the upright makes little difference. But I have always insisted upon a general anesthetic.

Dr. Edward Gray, Eldridge: I do not care to go into a discussion about anesthetics, but wish to discuss the status lymphaticus, the subject of the paper. Although the subject is new it has come before me in a practical way, and I wish for that reason to emphasize that portion of the Chairman's very valuable paper. He refers specifically to the psychic states as having influence. For that reason I think you will bear with me—because these things do not occur so very frequently—while I relate the history of a case which came before my notice just about one year ago. The patient was a feeble-minded individual 20 years of age. The feeble-mindedness was not of a very high order, but was accompanied by a partial paralysis so that the girl was unable to live outside of a chair. Emotional disturbances were well marked in that girl. For instance, it happened at least once or twice that when supper was brought to her and she didn't feel like taking it at that time and from that girl she got angry and threw the contents against the wall, or something of that kind.

The history of what happened relates only to 12 hours and I wish you to take note of this. What cause is there that can produce a fever that will rise in the course of three hours from an absolutely normal temperature to 105, to 106.5, and then fall to 103.4 and be followed by collapse and death in four hours? That is the history of a fair faced girl with a beautiful skin and auburn hair, who but for mental feebleness would have been a delight to look at. The history was simply this: This was an occasion when she was not feeling well. One of the girls under-

took to press upon the girl a certain portion of food. She flew in a passion and threw the food away. In an hour and a half the temperature went up over 104° and then up to 106°. I was called at 1 o'clock in the morning, and seeing she was in a desperate condition remained till four o'clock, and the uselessness of remedies was emphasized under such conditions. No amount of strychnia nor the more rapidly acting nitrite of amyl by inhalation produced a particle of effect, and about 5 o'clock in the morning, just about 12 hours from the seizure, she was a corpse. We had a postmortem. Going through the ordinary routine we were baffled completely, until I said to my assistant, "Let us look at the neck." Going to the neck we found a persistent thymus. That was my first clue. Then immediately we paid attention to the spleen. On section of the spleen you ought to have seen the Malpighian bodies stand out, as the American Reference Hand-book of the Medical Sciences says, "with vivid distinctness." Then there was hyperplasia of the large arteries, not at first suspected. So that the mechanism of that girl's death was the mental and psychic shock in the first place, which led to this paralysis afterward.

This case was reported in the American Journal of Clinical Medicine.

Dr. E. W. Fleming, Los Angeles: Under the head of the status lymphaticus Dr. Black has said the most trifling injury will at times produce fatal results. Now suppose we take an illustrative case. We have a patient 7 or 8 years of age who has enormously enlarged tonsils and obstructing pharyngeal tonsil, who has associated deafness as the result of hyperemia and swelling of the tube, and who has a moderate enlargement of the superficial glands of the neck, and now we will say just for argument that there is also a certain amount of what seems to be enlargement of the spleen. As I understood Dr. Black to say, it is impossible to determine beforehand that one is subject to the status lymphaticus. Now in that case, would you operate, and if you do operate what method would you follow? Would you give the patient an anesthetic or would you attempt to do the operation without one? Which would apparently be the safer course? I would like to put that question to Dr. Black.

Dr. J. H. McKeller, Pasadena: I have listened with a great deal of interest to Dr. Roberts' valuable paper and agree with

him on every point. I think a general anesthetic should be administered in children, and I think that it should be ether, or gas and ether. While working in the New York Eye and Ear Infirmary, for a period of about four months, the invariable anesthetic given was gas followed by ether. Not only was there no death, but I did not hear of one untoward symptom. It seems to me the great objection to the use of ether for tonsil and adenoid operations is the increased secretion of mucus in the throat which sometimes interferes with our work, but the greater safety more than makes up for that drawback. I have never seen bad results in adenoid operations under ether except in one case in the upright position. In my cases I have the patient in the recumbent position and always insist on ether being administered. I only remember having seen chloroform used once for the removal of tonsils and adenoids, and in that case the patient died shortly after beginning the anesthetic

Dr. Cullen F. Welty, San Francisco: I am not sufficiently familiar with the subject status lymphaticus to make any comment. I have operated cases that have had enlarged thyroids and just the other day I removed the adenoids and tonsils of a boy of about fourteen, who had a decided enlargement of the thyroid gland. I made careful observations of this case and there is nothing to report.

I am familiar with two deaths from chloroform anesthesia, one following curettment, and the other following the removal of tonsils and adenoids. I do not know the complete history of either case.

I never use chloroform at all because I consider it dangerous. I practically do all my tonsils and adenoids under ether anesthesia, and in the following way: Patient given a hypodermic of morphine and atropia, one half hour before operation. Primary anesthesia induced by nitrous oxide, and then supplemented by ether through Junker inhaler. Ether slightly warmed. In this way the patient gets only the minimum amount of the anesthetic.

I am confident that the administration of morphia and atropia reduces the amount of the anesthetic. Furthermore, the patients are much more quiet following the operation, and this is to be particularly wished for after complete enucleation of the tonsils.

Dr. F. D. Bullard, Los Angeles: What size doses?

Dr. Welty: Dependent upon the age and weight of the child. From one twelfth to one twentieth of a grain of morphine, one two-hundredth to one four-hundredth grain of atropia.

In regard to local anesthesia for enucleation of the tonsil in children, I will say that it is not satisfactory in my hands. They will not keep quiet sufficiently long to accomplish what I wish. In grown people I use one half per cent solution of cocaine, in one eight-hundredth adrenalin injection into the pillars. This is for small buried tonsils. The large tonsils I prefer to remove under general anesthesia.

I have come to believe, from our pathologic findings, that if you are going to operate upon a tonsil at all, it should be removed with the capsule, or in other words, a complete enucleation.

Dr. F. L. Rogers, Long Beach: I feel that I might with propriety add something to what the writer of the paper, and previous speakers have said, particularly from the fact that I hail from the city by the sea, where there have been reported three deaths, in fourteen months, from chloroform administration.

The first case reported by Dr. Roberts happened in my office, one year ago day before yesterday,—following the amputation of two very large middle turbinates, a double tonsillotomy and a probing of the frontal sinus. I gave the anesthetic myself and was assisted by my regular office attendant, an experienced graduate nurse. The patient a strong, well developed boy of thirteen was examined by me on two occasions, for heart or lung defects and pronounced normal. He had the usual preparation for operation, cathartic and no breakfast and took the anesthetic without fear or protest. Squibb's chloroform was used; less than one ounce was required to give complete relaxation. Anesthetic was withdrawn and the work completed rapidly; bleeding was moderately profuse, and pulse good at the time the operation was completed; and respiration not seriously interfered with up to that time. As I stood beside the operating table, with my hand on his pulse I felt a sudden weakening of the beat and noticed a death like pallor of the cheek; he was still breathing but it soon became intermittent and I realized there was serious trouble. Restoratives, artificial respiration, everything usually at hand were applied; two or three feeble gasps followed, but he never had a heart stroke we could feel after the first cessation of the pulse beat.

I desire to call attention to two lessons which this unpleasant experience of mine teaches, and to say that I believe it cannot be too often repeated: first, that no matter how simple the operation to be performed on a child, the consent of both parents to the

operation, either by word of mouth or writing, should be secured by the surgeon in advance, and then if possible have both within calling distance during the operation. In my case both parents were present and fully acquiesced in all that was done and exonerated me at the time and later both publicly and in private. I demanded that they do this, otherwise I would insist that an autopsy be held to further ascertain the cause of death. I have since regretted that a post-mortem was not held for the sake of demonstrating the presence or absence of the status lymphaticus.

Second, in minor surgery about the head, particularly in adenoids, where a quick general anesthetic is required, I have for many years occasionally administered my own anesthetic, usually chloroform, and depended on my own alertness, a deft hand, and a well trained office assistant to bring my patient through safely. But since this very unpleasant experience, the first of the kind in nearly twenty years' work, I have looked with disfavor upon the practice and shall, in the future, I think, be much less inclined to take the double responsibility, that is better shared with a colleague; even though arranging for an anesthetist often results in unpleasant delays and consequent embarrassment to patient and operator. The case had no adenoids and had never had a previous operation or anesthetic. This unhappy experience came to me on the anniversary of the San Francisco earthquake, which I passed through the year before and it jarred my nerves quite as much as that. I am not superstitious, but you will pardon me for declining to operate on a case day before yesterday, (the second anniversary of the quake) and for assigning as my reason, the fact that I wanted to attend this meeting.

Dr. S. P. Black, Pasadena: It is hard work to answer a hypothetical question, as is known by those who have had that experience on the witness stand. But in a case of that kind I think I should warn the parents of the child of the possible danger. I do not think we are ever justified in giving an anesthetic without giving a warning in any condition; but if they consented I should use ether if I gave a general anesthetic. I do not know how cocaine would work. As we know, cocaine produces a great many sudden deaths, and possibly these were cases of the status lymphaticus. I should not under any circumstances use chloroform or cocaine; eucaine possibly—it is on trial—or ether.

Dr. E. W. Fleming: Would you operate without an anesthetic?

Dr. Black: No.

Dr. Kasper Pischel, San Francisco: These exceedingly interesting reports show the truth of my statement before that operations done under a general anesthetic involve certain dangers. If I am correctly informed death several days after the operation from auto-intoxication is more frequent than during the operation. Therefore, I think if the family asks us, we have no right to say: "No possible harm can come from an operation." We cannot say it has nothing to do with the operation either. The family will not think so. We are not employed as mechanics to cut out a tonsil or chisel a hole into the skull, but we are called as physicians; great responsibilities rest upon us; therefore it is our duty to inform them that there are certain dangers involved.

Dr. W. H. Roberts, Pasadena (closing): One objection I would have to using local anesthetics in doing tonsil work is because in the vast majority of cases where we are called upon to remove diseased tonsils there are in addition enlarged adenoids, and while we can anesthetize the tonsillar area, we cannot anesthetize satisfactorily the adenoid area. I think the shock which the child sustains through having adenoids removed under a local anesthetic more than outbalances the shock they might have from a general anesthetic. I do not believe that we can produce sufficient anesthesia of the tonsillar area unless we inject the pillars. The little patients I see in my practice would not allow me to remove the tonsils under a local anesthetic, because when I remove the tonsils I do a complete enucleation, doing a dissection from the pillars and removing the tonsil within its capsule, with a cold wire snare. I operate invariably with the patient in the upright position, and generally by natural light. I have a north light in my operating room so that, except on very cloudy or foggy mornings, I never have to use artificial illumination. I always have the child thoroughly anesthetized before raising, and then maintain the anesthesia with a foot pump apparatus with a tube going through the nose. My anesthetist uses the Brophy apparatus, and it is very satisfactory. With this there are two bottles, so that none of the pure ether droplets can get into the child's throat after passing through the second bottle. The objection to ether is the abnormal amount of mucus secreted, but if we are anticipating trouble we can use a prelimi-

nary injection of atrophine, the amount to be regulated by the age of the child, and that eliminates that difficulty and also fortifies the child against shocks.

One other thing Dr. Black spoke of, and that is that there have been many sudden deaths from cocaine; more than physicians are willing to admit. I think cocaine is more dangerous than ether no matter how little may be used, and then with any local anesthetic there is the element of shock left, because it is almost impossible to render the operation absolutely painless under local anesthesia.

CHAIRMAN ROBERTS: This completes the program, but I understand Dr. Pischel wishes to show a new instrument, and if so, this is the time to present it.

DR. K. PISCHEL, San Francisco: In connection with this tonsil work I take the liberty of showing an instrument which in my hands helps me in tonsil work under a general anesthetic. I find difficulty in holding the tongue depressor properly. The tongue holder slips in all directions, and so I devised an instrument which I think avoids that. It is a tongue holder constructed like a tongue depressor with a blade to put under the tongue. Then the upper blade contains a fine needle so that the tongue is held quite firmly. At the same time you can move the tongue depressor to the right or left. If one wants to operate on the left tonsil the tongue depressor is moved more to the left, and the tongue can be pulled forward as is found necessary. I would be glad to show that around, and would like the gentlemen to try it. I am sure you can make some improvement on it. It is a rather rough instrument which might be improved yet. The blades do not come together entirely so that they do not pinch the tongue too much, but they come together to within about 1 cm.

DR. PISCHEL exhibited and demonstrated the instrument.

PAPER:

SURGERY OF THE FACIAL NERVE.*

JOSEPH C. BECK, M. D.

Aside from the report of five cases of facial hypoglossal anastomosis for the cure of permanent facial paralysis that I have performed, and the demonstration of a gross specimen removed post-mortem from one of these cases which had been cured for over a year, showing the perfect union of the two nerves, I have nothing original to offer in this line of work. Consequently the large part of my article is a repetition of what is already well known and on record. I have, however, written up this subject with one special purpose in view, namely: Inasmuch as the greatest number of facial paralyses are due to pathologic conditions within the temporal bone, more especially in the field of the otologist, or following his operative procedures to eradicate it, as, for instance, the radical mastoid, jugular bulb and labyrinthine operations, I have decided to bring this subject before you *in extenso*, compiling all that is practically known of the anatomy, physiology, pathology and treatment of the facial nerve.

Up to the present time, this subject is considered more particularly in general surgery, neurology, and internal medicine, but it evidently falls within the realm of the modern otologist.

It becomes frequently necessary to do a radical mastoid operation, in order to cure a facial paralysis, or if following such an operation one obtains a permanent facial paralysis, there is every reason why the otologist should correct this condition and not depend on a general surgeon to do so.

While the operation is very delicate and tedious, it is not difficult, and certainly the delicacy and tediousness of any operation are more

*This paper belongs with the Transactions of the Middle Section of the Society.

characteristic of the work of the otologist than that of the general surgeon.

The operations for the cure of facial paralysis have gained considerable favor in the past five years, so that at the present time we have a great deal of material at our disposal to study the results from various methods. Murphy's article on surgery of the nervous system compiles practically all the literature up to the present day, comprising thirty-four cases, and completely goes over the pathology of the peripheral nerves. I am, through the kindness of Dr. J. B. Murphy, using that part of his article practically verbatim.

In order to study the cure of facial paralysis, one should have a clear knowledge of the anatomy and function of the facial nerve, with all its associated branches. I have, therefore, prepared these cuts, which will also serve to show the relationship in studying the anastomosis of the various nerves with the facial. Figure 1 shows the course of the facial nerve from the beginning at the cortex (upper segment), traversing the internal capsule and associating or uniting with the nucleus of the facial nerve at the floor of the fourth ventricle. From thence we speak of the facial nerve proper, or the lower segment. The fibers, then, pass in a curve about the nucleus of the sixth nerve, making what is known as the knee, being then jointed by the eighth or auditory nerve, as two distinct trunks. These nerves have their exit from the brain at the junction of the pons and medulla. A third little nerve, known as the nerve of Wrisberg, or nervus intermediaris, is seen between these two trunks in their course towards the internal auditory meatus. At this latter point the two nerves are in a disinct relation; the auditory is posterior to the facial. They then pass into the internal auditory meatus until the aqueductus Fallopii is reached, when the facial leaves the auditory nerve and passes into this (Fallopian canal) across the roof of the middle ear cavity. Then it takes a turn backwards and downwards, so as to occupy the innermost part of the lower half of the posterior meatal wall; it then passes through the bony canal in the mastoid process until it reaches the stylo-mastoid foramen. Here it again takes a horizontal turn towards the posterior surface of the parotid gland, just below the cartilaginous external auditory meatus, piercing the capsule of this gland, and entering the substance of it.

The nerve then divides at first into two branches, one of these again giving off a third. These three branches leave the gland, giving off a number of small branches, which pass to their respective muscles, anastomosing with the sensory nerve of the face, forehead and neck.

The various branches as they are given off, also branches associating or communicating with other nerves, are as follows:

Interosseous Branches:

1. Great superficial petrosal nerve, which communicates with the fifth.
2. Anastomotic branch with tympanic plexus.
3. Stapedius nerve.
4. Chorda tympani.

After Leaving Foramen:

1. Posterior auricular nerve.
2. Digastricus nerve.
3. Stylohyoideus nerve.
4. Communicating with the glosso-pharyngeal.

Parotid Plexus:

1. Ramus coli.
2. Mandibular ramus.
3. Buccal ramus.
4. Zygomatic ramus.
5. Temporal ramus.

FUNCTION OF THE FACIAL NERVE.

The facial is the motor nerve of all the muscles of the face, including the platysma, but not including any of the muscles of mastication. It supplies the parotid gland, and through its communication by means of the petrosal with the vidian, some of the muscles of the soft palate, probably the levator palati and azygos uvula (denied by some). By its tympanic branches it supplies the stapedius; through the otic ganglion the tensor tympani; through the chorda tympani it sends branches to the submaxillary glands, and to some muscle fibers of the tongue, through its connection with the lingualis. It supplies the mucous membrane, of the anterior two-thirds of the tongue. By some of its branches before it comes upon the face, it supplies the muscles of the external ear, the posterior belly of the digastric, and the stylohyoideus. It therefore, is a secretory nerve, as well as a nerve of special sense, aside from

its motor functions. The sense of hearing is also affected by the muscles which it supplies, as the stapedius and tensor tympani. Again, the sense of smell is somewhat controlled by this nerve, in that it permits proper dilatation of the alae nasi; in this manner a proper amount of air is inspired. The sense of taste is supplied by the chorda tympani to the anterior two-thirds of the tongue. Some observers claim that this branch is an entirely independent nerve, only accompanying the facial, but having its origin from the nucleus of the fifth. Sight is influenced by its action on the lids.

The surgical anatomy will be described in connection with the operations.

CAUSES OF FACIAL PARALYSIS.

These will depend a great deal on the different portions of the nerve involved; for instance, the intracranial, the interosseous, or the external portion. However, one may have a paralysis due to the involvement of two of the portions, as an ascending or descending neuritis. Enumerating some of the most frequent causes we have, first, lesions, as:

1. Hemorrhages, subcortical, in the internal capsule, and before the communication with the nucleus.

2. Brain abscess.

3. Gumma.

4. Tumor.

5. Meningitis

6. Suppurating disease of the middle ear, the most frequent cause, in the interosseous portion. It is most frequently in the region of the Fallopian canal, although it may occur anywhere along its course. Frequently it is associated with Bezold's mastoid abscess.

7. Traumatic, as following the mastoid operation (radical), or labyrinthine exsection. It sometimes follows pressure from gauze packing, or improper manipulation of the protectors for the facial nerve. Fractures of the base of the skull are at times followed by facial paralysis, or gunshot wounds of the temporal bone.

In the external portion we find it associated with:

8. Tumors of the neck.

9. Retromaxillary tumors or abscesses.

10. Following resection of glands of the neck.

11. Osteoperiostitis of the lower jaw.

12. Tumors and abscesses in the parotid gland, and operations for these conditions.

13. Trauma, as stab wounds or the characteristic gashes of the German duelists, causes a goodly number of external facial paralyses.

The general systemic diseases that cause facial paralysis are:

14. Syphilis.

15. Rheumatism.

16. Other acute infectious diseases.

Among the largest group of cases are those spoken of as Bell's palsy, which are supposed to be due to:

17. Exposure to cold and draught.

18. Toxic.

According to others:

19. A severe gastro-intestinal toxemia may cause facial paralysis.

Such local causes as:

20. Very strong remedies poured into the ear, or applied, as chromic acid, trichloracetic acid, nitrate of silver, have been reported as causing paralysis.

21. Excessively hot or cold instillations into the ear with perforating drum membranes are given as causes of paralysis.

HISTOLOGY OF THE FACIAL NERVE.

A cross-section of the trunk of the nerve shows, as any other peripheral nerve, the following structures: (Fig. 2.)

Section (transverse) of the nerve trunk shows in the first place bundles of nerve fibres (funiculi) ensheathed by a fibrous capsule (epineurium). Each bundle is surrounded by its own sheath known as thé perineurium. Each nerve fibre is surrounded by a sheath, known as endoneurium. Between the structures are seen lymph spaces, veins, arteries, fat.

The termination of the nerve fibres is by the muscle plates, which are the axis-cylinders breaking up into a number of cells, which are practically fused with the sarcolemma of the muscle.

Section of Nerve Fibres (fig. 3):

(1) External (primitive nerve sheath) nucleated sheath of Schwann, or neurilemma.

(2) Intermediate or packing substance, also known as medullary or myeline sheath, or white substance of Schwann.

(3) Internally the axis-cylinder, otherwise known as primitive band, axis band, or axial fibre.

The neurilemma is structureless, and within it are seen at intervals nuclei, surrounded by protoplasm.

The medullary substance is supposed to be made up of two distinct parts: (a) a reticular framework; (b) fatty substance of a semi-fluid consistency, staining readily with osmic acid. The reticular network, according to some authors, has a highly chromatic property which is very resistant to chemical and digestive fluids, known as the neuro-keratin network.

The axis-cylinder consists of primitive fibrillae and can be traced from origin to termination.

Nodes of Ranvier. At regular intervals in the nucleated sheath of Schwann one can see a constriction. At these points the external sheath and axis-cylinder come in immediate contact. Between each two nodes of Ranvier is what is termed an internode. The latter varies in size, but averages about 1 mm. Only one nerve nucleus to each internode can be demonstrated. The constricting band of Ranvier is found between two internodes, uniting them to the external sheath, which band stains black with silver nitrate. Since the axis-cylinder also stains with silver nitrate, one can therefore at each node of Ranvier see a cross known as cross of Ranvier.

The size of a nerve fibre varies much in its course, the average being 6.2 to 8.7 micra.

PATHOLOGY OF THE FACIAL NERVE.

We may have either (a) neuritis or (b) death of the nerve. In the case of the former there is an increase in the vascular and plasmodic supply, with swelling of the component parts of the perinurium. There occur a round-cell infiltration and naturally a compression of the axis-cylinder. The degree of inflammation varies and in the marked cases may terminate in permanent destruction of the nerve, that is, there may be gangrene or suppuration.

In the case of severance of the continuity of structure of the nerve, either by being cut or injured, as in fractures or gunshot wounds, in association with and complication of a suppurative process of the temporal bone, or tumors involving the nerve cell, one will see the following changes: According to F. Marchand,[1] the whole peripheral end of the nerve degenerates, as well as a small part of the central, up to the first node of Ranvier. On the second day after the division, the medullary sheath breaks down in the

form of cylindrical fragments, which continue subdividing during the next few days. These fragments are still nucleated in the sheath of Schwann. The axis-cylinders also fragment, and are either swollen or fibrillary in aspect. They are included among the myelin fragments. At the same time a regenerative process begins in the nuclei of the sheath of Schwann; the nuclei enlarge, become richer in chromatin, and penetrate between the fragments of the myelin. Simultaneously, the protoplasm increases in volume and fills up the empty spaces between the drops of myelin. The latter are surrounded by protoplasm and form around nucleated lumps. The nuclei of the sheath multiply by karyokinesis on the third and fourth days, and subsequently become very abundant in numbers. The decayed medullary sheath and axis-cylinders, which are in the form of small, round drops, are often mistaken for fat. A great part of these nuclei, surrounded by protoplasm, transform these cells into long spindle elements, the importance of which is variously interpreted. This feature will be considered when the process of regeneration is taken up.

According to Ballance and Stewart,[3] the various steps in the process of degeneration are as follows: (1) Cellular change; (2) changes in the axis-cylinder and medullary sheath.

Cellular Changes.—The first sixteen or eighteen hours after the division of the nerve there is a diapedesis of leucocytes in the entire distal segment, and also in a small portion of the proximal segment. In from three to fourteen days, connective tissue cells replace these leucocytes. Similar to the invasion of the nerve with the leucocytes on the second day after the injury, there is a marked proliferation of the connective tissue cells, invading the entire distal segment and a small portion of the proximal. On the fourth day the fatty substance of the myelin fragments is included within the substance of the connective tissue cells. The exact time in which the absorption is completed has not been determined, according to these authors. It is estimated, however, that the myelin disappears in about five weeks. After the complete absorption of the myelin, the connective tissue cells diminish and are outnumbered by the gradually increasing neurilemma cells. The remaining connective tissue cells become spindle-shaped and their destination is the formation of connective tissue fibres separating the chains of neurilemma cells. On the second day after the injury the neurilemma cells proliferate. At first their proliferating activity is greater than that of the con-

nective tissue cells, and during this period they exert an absorptive action upon the degenerating medullary sheath. Later, their proliferating activity is surpassed by the connective tissue cells, the former continuing to proliferate in columns. The young neurilemma cells preserve the original longitudinal direction of their parent cells, and from their opposite poles they shoot out fine protoplasmic processes which gradually increase.

Changes in the Medullary Sheath.—Decay of the axis-cylinders begins on the fourth day, and is very pronounced on the seventh. Fine fibres have a greater resistance to disintegration than the coarse ones. The fragments of the axis-cylinders are absorbed by the connective tissue cells principally, and partially by the neurilemma cells. The degenerative process is not influenced by immediate suture, as it takes place uniformly. The muscle supplied by the divided nerve rapidly diminishes in weight.

Regeneration.—This begins, according to Ballance and Stewart, before degeneration is completed. At the end of the third week the peripheral segment contains a great number of elongated spindle cells, which shoot out axis-cylinders from their poles. The scar tissue contains cells whose ends interlace in an irregular manner, and form a new network of new axis-cylinders. The anastomosis of the axis-cylinder of the two ends of the nerve is accomplished at the end of four weeks.

SYMPTOMS.

These will depend on the location of the lesion, with reference to the involvement of other nerves or parts of the body. There is, however, a characteristic picture of a facial paralysis that can not be mistaken very well for any other condition, although we may have different degrees of paralysis, as a mild paresis or complete paralysis.

The characteristic picture (Fig. 4) shows the angle of the mouth drooped, the usual nasolabial fold obliterated; in fact, the whole cheek hangs flabby. The lower eyelid sags and the upper one lies closer to the eyeball; the eyebrow seems lowered as well as the skin covering the forehead—the usual creases of this region are wanting. Tears are found gathered in the corners of the eyes, and often seen trickling over the lower lid. The bulbar conjunctiva is frequently injected. So far as the patient is concerned, there is a most unpleasant appearance and sound when he begins to speak, to laugh or attempts to move the facial muscles. He can

not close the eye, and when forced to do so, the eyeball rolls up, and one sees only the scleral portion. Asked to whistle or to blow up the cheeks, one finds that it is impossible for him to do so. The air will escape through the paralyzed side of the mouth. Patients complain a great deal of their food lodging within their cheeks, and find considerable difficulty in clearing the buccal surfaces. They generally use the other side of the mouth for mastication. Inasmuch as most of the cases are those where the nerve is injured above the chorda tympani, there are symptoms of perverted or loss of taste. A complaint frequently made is that of a metallic taste. The eye symptoms bother the patient a great deal, especially when exposed to the wind, and on first arising in the morning.

PROGNOSIS

This depends absolutely upon the cause and location of the lesion. Suffice it to say, however, that in general the prognosis is bad and the course is a long and protracted one, except in that group of cases known as Bell's palsy, such as are classed as toxic neuritis, rheumatic, luetic or compressive. In the latter cases, when the condition is relieved, recovery takes place more readily, and even here one must give a very guarded prognosis. I have had a number of cases of the above type that resisted all sorts of local and general treatment, and only after three years did complete recovery take place. However, most of them recovered much sooner, between three weeks and six months.

Again, the prognosis depends on the treatment. I mean, specifically, surgical treatment, and when instituted. To be more specific, if one has a complete paralysis, with a positive reaction of degeneration, it is best not to wait too long before operating for fear of losing the muscular action, as the muscles tend rapidly to undergo trophic changes and waste away, after which an operation on the nerve will be practically useless. Another important point is the onset of paralysis, especially after operation. If it be gradual and take two or more days before development, the progress is better than if it has occurred as soon as patient comes out of the anesthetic, which condition is more grave.

REACTION OF DEGENERATION.

Normally, the facial, like other motor nerves, is affected by electrical stimulation as follows: The faradic current, when applied over the trunk of the facial, behind the ear or over the point

of emergence of its branches on the face, causes a quick contraction of muscles supplied by these nerves. The electrodes may be applied directly over individual muscles with the same result. When a sufficient amount of galvanic current is applied over the nerve or muscles, contraction occurs every time contact is made, with the so-called "make" of the current; slightly stronger currents produce another contraction when the current is released or "broken." In all these stimulations the "cathode" or negative pole has been found to be the more active; that is, less current is required to get a contraction when the negative pole is placed upon the muscle to be stimulated; while the positive electrode may be put upon an indifferent point, preferably the sternum or back of the neck. The normal formula usually given is:

C. C. C.>A. C. C.=A. O. C.=C. O. C.

In which C. stands for cathode, A. for anode, C. for closing, C. for contraction, O. for opening.

C. C. C.,cathode, closing contraction.

>A. C. C., greater than anode closing contraction.

What do we mean by the reaction of degeneration, which is abbreviated by R. D.?

In this, the important points are that faradism fails to produce a contraction both through muscles and nerves, while galvanic currents produce no contraction of the muscles when applied over the nerve, but still cause muscular contraction when applied directly over the individual muscle. There is, however, a change in the character of the contraction. It has ceased to be prompt, lightning-like, but has become sluggish, peristaltic-like. A minor point is the inversion of the polar reactions. Whereas in the normal muscle the cathode is the more active in closing or making of the current, in the degenerated muscle the anode may be more active in closing and less than normal in the opening contraction, that is, the reverse of the normal muscle. This change, however, is unimportant. The time of occurrence of the reaction of degeneration is from eight to ten days following injury or disease of the facial nerve.

Immediately following injury, the nerve response may be even greater than normal; that is, less current is required to produce the effect. Authors have differentiated various degrees of reaction of degeneration according to the completeness or incompleteness of the reaction formula. The prognosis, therefore, is somewhat governed by the fact as to whether reaction of degeneration is

complete or incomplete. The less complete the R. D., the better the prognosis.

TREATMENT.

The treatment must necessarily again depend on the etiology and pathology, as well as on the location of the lesion. In a general way, however, we can say the treatment is either expectant, local, general, medicinal, mechanical, or surgical. Taking up the cases systematically, as mentioned in the etiology, I will state that the local treatment will constitute in the first place, removing, if possible, whatever may be pressing on the nerve, as tumors, abscesses, necrotic bones, sequestra, and other foreign bodies. One of the frequent procedures necessary is a mastoid operation, and the removal of cholesteatomatous masses, sequestra and granulations over the region of the facial canal. Quite a number of cases of this sort will get well after such procedure.

Facial massage, vibratory massage, etc., may be systematically carried out, twice or three times daily for a half an hour at a sitting.

Electricity—a mild faradic current, is to be used as follows: The positive pole is applied over the sternum, and the negative used as the stroking electrode over the whole paralyzed area, once daily for three to five minutes, followed by galvanic current, the technic practically the same, using from five to ten milliamperes for three to five minutes.

The eye must be protected from external influences, as foreign bodies, dust and wind will irritate the condition; usually I give the patient a shield or a large lens (plain). Over night the eye should be tied up. A drop of argyrol, 5 per cent, and boracic acid irrigations are good adjuvants to prevent infection.

Thorough cleansing of the mouth and especially the inner surface of the cheek of the paralyzed side, as food particles remaining therein produce considerable irritation from decomposition.

The general treatment consists in tonics, particularly strychnin sulphate, 1/60 of a grain three times a day.

Eliminating and reabsorptive remedies should be used, as cathartics, sweat baths, potassium iodid solution.

A very wholesome nutritious diet, such as will leave the least residue, is to be advocated; rich condiments and red meats are to be restricted.

Strict attention to hygiene and exercise is to be encouraged, particularly in the open air. A change of scene is to be recom-

mended, that is, away from friends and acquaintances, because patients are very frequently sensitive about these conditions, and very often become morose, this condition causing delay in the recovery.

Of course, if it be a case of luetic facial paralysis, a thorough anti-syphilitic treatment by mercury and iodid of potash is to be instituted, associated with all the other general and local treatments required in that condition.

SURGICAL TREATMENT.

The indications for an operation on the facial nerve are confined to absolute paralysis, no matter from what cause and wherever the location of the lesion (lower segment), after all other methods have failed. The procedures are usually anastomosis with other neighboring motor nerves, as hypoglossal, spinal accessory and glossopharyngeal, in the order named.

The contraindications are: (1) When the general condition of the patient will not warrant such an extensive operation, and (2) when the other nerves, as hypoglossal, spinal accessory and glossopharyngeal, are not in good condition or accessible. (3) In case of congenital absence of the facial muscles or nerve. (4) In long standing facial paralysis where one, in making the test of reaction of degeneration, can safely say that all the muscles supplied by the facial nerve are wasted and beyond the possible chance of regeneration. This last contraindication must, however, be modified inasmuch as there are cases on record (Hackenbruch) in which the above conditions existed, which were operated upon and recovered.

The historical data of neuroplasty may be briefly stated as follows: Probably Ballance and Stewart were the first to have united the facial and spinal accessory nerves for the cure of facial paralysis, in 1895, but did not publish it until 1903.[4] In 1898, at the suggestio nof Fouret, Foure[5] united the facial with the trapezius branch of the spinal accessory. Experimentally, P. Manasse,[6] in 1900, and about the same time Barrago Ciarello[7] accomplished satisfactory results on animals in uniting the facial with the spinal accessory. Robert Kennedy,[7] in 1900, operated on a man by uniting the facial with the spinal accessory. Harvey Cushing[8] performed the same operation in 1902. After that, quite a number of similar operations followed. The hypoglossal, however, was not used until about 1902-3, as cases reported by Ballance and Stewart (*British Med. Journal*, May, 1903). Koerte,[9] Nicoll,[10] Frazer,[11] Bardenhauer,[12]

Schaefer,[1] of Edinburgh, suggested the union of facial and glosso-pharyngeal.

Herewith follow the cases tabulated by Murphy, with some additions by myself from literature and personal communication as well as cases operated on by myself:

NERVE ANASTOMOSIS FOR THE CURE OF FACIAL PARALYSIS, TIC, OR SPASM.

A. Experimental.

Case 1.—Paul Manasse. (Munk's Laboratory.) (*Arch. f. klin. Chirurgie,* vol. lxii, p. 822.) Seven dogs operated on, but only five considered in final results. Spinal accessory freed from its implantation into sternomastoid muscle to atlas; end-to-end anastomosis of central spinal accessory with peripheral facial divided at the stylomastoid foramen. Four silk sutures passed through quarter of the thickness of spinal accessory.

Results.—Clinical results not sufficiently satisfactory. In first three months there was no faradic response; the latter obtainable after four months. Electric stimulation of central spinal accessory was followed by response in peripheral facial. Histologically, new axones penetrated the facial from the central spinal accessory. Manasse thinks that "months" is an insufficient time for voluntary motion and disappearance of atrophy. "Years" would lead to perfect restoration.

Case 2.—Barrago Ciarello. (*Policlinico,* 1901, No. 3.) In two dogs. (a) Anastomosis between central end of spinal accessory and peripheral of facial. (b) Anastomosis between the central end of vagus and peripheral end of facial.

Results.—(a) Reformation of function commenced after four months, and was almost completely restored in six. There was still some muscular atrophy left. Reaction of degeneration disappeared. (b) Same results. Ciarello believes that spino-facial and vagofacial anastomosis is promising in man in recent facial paralysis.

B. Clinical.

Case 1.—Charles A. Ballance, Hamilton Ballance, and Purves Stewart. (*Brit. Med. Jour.,* May 2, 1903.) Facial paralysis. Following operation for suppurating mastoiditis. Spinofacial anastomosis. (Operation by C. A. Ballance.)

Results.—After several years, muscles responded to galvanic and faradic currents. Still some drooping of angle of mouth and widening of palpebral aperture. Movements always associated; voluntary independent movements of muscles of face not possible.

Case 2.—Ballance and Stewart. Facial paralysis. Otorrhea of twenty years' standing. Spinofacial anastomosis performed. (Operation by C. A. Ballance.)

Result.—Some improvement after ten months.

Case 3.—Ballance and Stewart. Facial paralysis. Following operation for otitis media. Spinofacial anastomosis performed after six months). Anastomosis of central end of spinal accessory with side of facial. (Operation by H. Ballance.)

Results.—All muscles of face responded to faradic currents after four weeks. Three months later, some voluntary motion. Three sutures of French silk used.

Case 4.—Ballance and Stewart. Facial paralysis. Following operation for otitis media. Spinofacial anastomosis, four months after the initial paralysis. (Operation by C. A. Ballance.)

Results.—Faradic response after nine months. Associated movements only. After twenty-one months, voluntary independent elevation of inner end of eyebrow.

Case 5.—Ballance and Stewart. Facial paralysis. Fracture of skull, due to a fall of fifteen feet. After thirty-five months. Spinofacial anastomosis. (Operation by C. A. Ballance.)

Results.—Slight voluntary movements in muscles of face if shoulder was elevated under resistance.

Case 6.—Ballance and Stewart. Facial paralysis. Following operation for otitis media. Spinofacial anastomosis six months after initial paralysis. (Operation by C. A. Ballance.)

Results.—No improvement; no electric response; no return of independent voluntary or associated movements.

Case 7.—Ballance and Stewart. Final paralysis. Following the removal of a cystic tumor of lower jaw. Operation twelve months after initial paralysis. (By C. A. Ballance.) Anastomosis of peripheral end of facial with side of hypoglossal.

Results.—No improvement at the time of operation. Atrophy of half of the tongue.

Case 8.—Faure. January 28, 1898. (a) *Gazette des Hopitaux,* 71 eme annee, 1898, p. 259. (b) *Revue de Chir.,* 1898, t. xviii, p.

1008. Facial paralysis. Injury. Eighteen months later, anastomosis of facial with accessories.

Results.—Slight improvement some time after the operation. "Half successful."

Case 9.—Robert Kennedy. Phil. Trans. of the Royal Society, series B, vol. cxciv, p. 127. Facial tic (facial hemispasm). Spinofacial anastomosis. End to side.

Results.—Cured. After 470 days, patient able to close eye. Muscles regained volume, and contracted voluntarily.

Case 10.—Hackenbruch (Wiesbaden). *Verhandl. der deutschen Gesellschaft f. Chir.*, 1903, bd. xxxii, p. 231. Facial paralysis. Sudden paralysis of facial, three months' standing. Seven and three-fourths years after appearance of paralysis. Spinofacial anastomosis.

Results.—Improvement after four and one-half months. After nine months, patient could draw the angle of the mouth downward.

Case 11.—Korte. (*Deutsche med. Woch.*, April 23, 1903.) Facial paralysis. Following operation for ostitis of mastoid process. Hypoglosso-facial anastomosis.

Results.—Good after one and one-half years, but associated movements present.

Case 12.—W. W. Keen. Phila. Neurol, Soc., Feb. 24, 1903. (*Jour. of Nerv. and Ment. Dis.*, Feb. 24, 1903, p. 366.) Facial paralysis. Struck by a glass bottle. Five weeks after accident, Spinofacial anastomosis.

Results.—"Perfectly smooth recovery."

Case 13.—Harvey Cushing. (*Ann. of Surg.*, May, 1903.) Facial paralysis. Revolver-shot behind ear, involving mastoid. Operation forty-three days later. End-to-end spinofacial anastomosis.

Results.—Complete restoration of function.

Case 14.—Hackenbruck, (*Arch. f. klin. Chir.*, 1903, Bd. lxxi, p. 631.) Facial paralysis of seven years' and nine months' standing. Spinofacial anastomosis by implanting two-thirds of spinal accessory into facial.

Results.—Electrical response in paralyzed muscles. Voluntary contraction independent of those of trapezius.

Case 15.—James H. Nicoll. (*Lancet*, 1903, p. 956.) Convulsive tic. Anastomosis of facial with hypoglossal.

Results.—No improvement at the time of communication.

Case 16.—Szeyner. (Abstracted in *Fortschritt der Mediz.*, 1903, p. 1060.) Facial paralysis. Shot; attempt at suicide. Facial anastomosis with a flap taken from spinal accessory.

Results.—Voluntary contractions after three months; able to close eye, and whistle.

Case 17.—Charles H. Frazier. (*Univ. of Pa. Med. Bull.*, 1903-4, vol. xvi, p. 306.) Facial paralysis. Gunshot wound. End-to-side anastomosis between peripheral facial and side of hypoglossal.

Results.—Too recent to report conclusively on the case.

Case 18.—Glück. (*Berl. Gesell. f. Psychiatrie*, May 11, 1903.) Facial paralysis. Division of facial during mastoid operation for otitis media. End-to-side anastomosis between peripheral facial and side of spinal accessory.

Results.—Regeneration of facial. Associated movements.

Case 19.—A. I. Bouffleur, Chicago. Personal communication. Facial paralysis due to gunshot wound (attempt at suicide), June 14, 1904. The tip of mastoid was cut off by the bullet. Division of lower branches as well as of common trunk of facial. End-to-end anastomosis between spinal accessory and facial, performed four weeks after injury.

Result.—Case lost sight of.

Case 20.—Dr. O. Facial paralysis. Following operation for otitis media. March 10, 1904. End-to-end spinofacial anastomosis.

Results.—Muscular atrophy not improved. Patient has associated movements only in muscles of face.

Same case as above. J. B. Murphy. Facial paralysis. Following operation for otitis media. March 10, 1906. Freed nerve from scar tissue and imbedded it in muscle.

Results.—Muscular atrophy improved, and voluntary motion of face is manifest. The associated movements continue. Patient was reoperated on by the author.[1]

[1] The point of anastomosis found surrounded by great mass of connective tissue.

Electrical irritation of central end of spinal accessory produced contractions in muscles of the face, showing that new fibers had grown from the spinal accessory into facial, but that impulses could not travel, on account of surrounding connective tissue. Point of anastomosis was freed and protected by soft tissues. Good results.

Case 21.—Loehlein. (Communication, *Vereinigung der Chirurgen*, Berlin, July, 1904.) Facial paralysis. Otitis media. Anastomosis of facial with spinal accessory.

Results.—"Excellent results." No associated movements.

Case 22.—Bardenheuer, (*Munch. med. Wochenschr.*, 1904. No. 28.) Facial paralysis of ten years' standing. End-to-end anastomosis between peripheral facial and side of hypoglossal.

Results.—"Very good." Tongue paralyzed. No associated movements.

Case 23.—Bardenheuer and Lambeth, (*Festschrift zuh Eroffnung der Akademie f. praktische Medicin zu Koeln*, 1904, p. 219.) Facial paralysis. End-to-end anastomosis between peripheral facial and side of hypoglossal.

Results.—Good.

Case 24.—Mintz. (*Zentralbl. f. Chir.*, 1904, No. 22, p. 684). Facial paralysis. Trauma during mastoid operation for otitis media. Anastomosis of facial with spinal accessory.

Results.—Complete restoration of motor function. Good closure of eye.

Case 25.—Villar. (*Gaz. hebd. des Sc. Med.*, Bordeaux, Feb. 7, 1904.) Facial paralysis. Trauma to facial during mastoid operation for otitis media. Anastomosis of facial with spinal accessory.

Results.—"The results were astonishing, as patient was able to move his lid on next day after operation. This rapid improvement, however, did not remain permanent."

Case 26.—Alexander. (*Wien. med. Wochenschrift*, No. 11. 1904.) Facial paralysis. Otitis media paralysis of five years' standing. End-to-side anastomosis between divided facial and side of hypoglossal.

Results.—Paralysis of half tongue. No improvement at time of communication.

Case 27.—Alfred S. Taylor and L. Pierce Clark. (*Jour. Am. Med. Assoc.*, March 24, 1906.) Facial paralysis. Suppurative mastoiditis. The peripheral segment of facial inserted into a slit of hypoglossal.

Results.—Improvement after twenty-five months. The anastomosis was protected by Cargile membrane.

Case 28.—Same. Facial paralysis. Paralysis followed a severe earache. Same operation.

Results.—Improvement.

Case 29.—Same. Facial paralysis. Suppurative mastoiditis. Same operation.

Result.—Improvement.

Case 30.—Same. Facial paralysis. Bell's palsy. (Paralysis twelve years' standing.) Same operation.

Results.—Improvement. Although paralysis existed for twelve years, facial nerve was normal in size and aspect.

Case 31.—Same. Facial paralysis. Suppurative mastoiditis. Same operation.

Result.—No improvement.

Case 32.—A. I. Bouffleur, Chicago. Personal communication. Facial paralysis resulting from fracture of mastoid, concomitant with fracture of skull (1903). Nerve-trunk intact, as far as continuity is concerned. End-to-end anastomosis between spinal accessory and facial. Performed September 11, 1906.

Results.—Some improvement shortly after operation, inasmuch as fluids did not run out of the mouth from paralyzed side. Time too early to give definite results.

Case 33.—J. B. Murphy. Present paper. Facial paralysis. Division of facial during mastoid operation for otitis media. End-to-end anastomosis between peripheral facial and central spinal accessory.

Results.—Evidence of returning motion pronounced. See history. Too recent for complete recovery.

Spiller, University of Penn., *Med. Record,* November, 1903.

Rothmann, *Berliner klin. Woch.,* 1903, No. 34.

Bernhardt, *Berliner klin. Woch.,* 1903, No. 34.

Koster, *Berliner klin. Woch.,* 1903, No. 34.

Gradenigo, Laryngoscope, 1907.

McArthur, Eisendrath, Ochsner, Chicago surgeons, have done this operation, but for some reason have not reported their results.

Beck, J. C., five cases of facial hypoglossal anastomosis. (Present paper.)

GENERAL POINTS AS TO NERVE ANASTOMOSIS.

In the first place, a clean, aseptic field for primary union of the sutured ends is practically necessary to obtain rapid regeneration through the anastomosed ends; proper protection (as shown in Murphy's cases) of the anastomosed ends, to prevent access of cicatricial formation about them, by placing into a neighboring muscle; absolute rest to the parts, almost immobilization of the

head for the first few days after operation, to prevent severance or traction on the united ends.

The methods of anastomosis most frequently employed are:

1. End to end.
2. End to side.

The most preferable mode is that of implanting the exposed stump on the paralyzed side into a niche of the living nerve; this niche should have its axones about 1-2 to 2-3 cut transversely, in order to obtain direct contact and union, and, secondly, so that absolute loss of function does not result in the muscles supplied by the live nerve, as, for instance, the tongue would only be partially paralyzed when the hypoglossal is used.

SUTURE OF NERVES.

Either primary or secondary. By "primary suture" is understood suture of the two divided ends immediately after the accident or within the first twenty-four hours. Secondary suture is the approximation of the two divided ends within several months or years after the injury. The older surgeons base their practice on the theory that a peripheral segment which has been disconnected from the central for a long period of time is permanently degenerated, and an attempt at union would be futile. The most modern surgeons, better informed on the histology and physiology of the peripheral nerves, and especially those who believe in peripheral regeneration, are of the opinion that new nerve fibers regenerate in the peripheral segment, even in it has remained disconnected for a considerable period of time. The fibers are imperfect, and as soon as they are united with the central end, under the influence of central impulses they develop to anatomic perfection and to normal physiologic function. The limitations of time in which this restoration may take place has not been definitely determined, but restoration of function has been secured many years after division.

In any operation upon the nerve trunks, one must, first of all, identify the nerve; otherwise the operator is in the dark, and the final result will undoubtedly be compromised. This identification can easily be accomplished by making use of the nerve excitor. This is composed of a metallic cylinder which can be attached at one end with the poles of a faradic battery, while the other end is provided with two platinum needles or wires, three centimeters in length, and situated a distance of half a centimeter from each other. These tips can be easily sterilized, and should always be at

hand when operating upon the nerve trunks. The importance and the value of this instrument can be appreciated when we think of the conditions of the tissues in an old and extensive injury in which they are all matted and bound together by connective tissue. The structures have lost their natural color, and the anatomic relations and identification of nerve trunks becomes an impossibility without the use of the excitor.

Operative Procedure.—The first essential is the removal of all connective tissue that caps the nerve endings to a degree sufficient to expose clearly the fasciculi and neurones, even though this shortens the nerve beyond the possibility of direct end to end suture. The axis-cylinders or nerve fibers will span a considerable distance under favorable conditions, but they will not penetrate connective tissue masses, so that it is absolutely necessary to have all the cicatricial or new-formed connective tissue removed from the nerve ends. The failure of this removal has contributed more than any other element to the failure of restoration of function after nerve suture. This, of course, has reference to secondary suture.

The needle used for suturing should be a round intestinal needle, without cutting edges. Suture material should be fine linen, silk, kangaroo tendon, or slowly absorbable catgut. The suture should not be passed through the substance of the nerve, but through the perineurium, and also through the surrounding connective tissue. This point is very essential for success. The trauma produced by passing the needle through the substance of the nerve, and especially if a slight infection takes place, causes a proliferation of connective tissue which compromises the restoration of function. Figure 5 shows the various methods of suture.

If the nerves face each other, and are not separated by a considerable gap, a slight traction established when the muscles are completely relaxed, will make it possible easily to approximate the ends. A little stretching of the ends can be easily made without any prejudice to the ultimate result. If the distance is considerable between the two ends, the nerves can be united in several ways. Letievant is the originator of "suture a lambeaux," or flap-suture. Foreign bodies, nerve segments from other animals, portions of segments of spinal cord from small animals, have been interposed between distant segments. In the last ten years, in cases of considerable distance btween ends, the divided peripheral end was implanted into a neighboring healthy nerve, which was only partially

divided. In several instances, especially where the trauma has compromised the nerve and bone, in order to be able to approximate the two ends a segment of bone was resected, that the divided ends might be brought in contact (Keen, Trendelenburg). Bungner interposed a piece of human brachial artery between the segments of the peripheral nerve in a dog. After 43 days the space between the two divided ends of the sciatic was filled with new nerve fibers. Bungner ascribed to the artery the role of a director and medium along which the nerve fibers can travel in a straight direction. Nothaft interposed a segment of rabbit's aorta between the two ends of the sciatic, from which he had excised one-half centimeter. When the gap between two ends is considerable, the contact can be established by foreign material. In a case of injury to the radial, Gluck, in 1888, interposed catgut bundles between the ends, which were separated by a gap of 5 cm. The gap was filled in six months, or, as he says, there was a neurotization of the catgut bundles. The result was good.

On the strength of Forsman's observation on "positive neurotropism," several surgeons and experimenters have inserted the two ends into a tube of decalcified bone, previously sterilized; this has enabled a more rapid union of nerve fibers.

"Suture a Distance."—This procedure originated with Assaky (Roumania). By this is meant the interposition of catgut fibers between the two ends of a divided nerve where a considerable gap exists. In Assaky's experiments (4 dogs, 2 rabbits), in the dogs favorable results were obtained in from 67 days to 4 months. The greatest segment successfully excised was 3 cm. Once he resected 6 cm. and interposed catgut fibers between the ends; no regeneration occurred after 39 days. If the distance is considerable between the segments, a piece of muscle may be interposed, or the two segments may be imbedded in a muscle from the vicinity in such a manner as to place the cut surface of each end face to face. This has been done by Levings, of Milwaukee, and several times by Murphy. The ideal results in nerve suture are obtained, as in the repair of all other tissues, by an exact juxtaposition of similar histologic elements, end to end. This requires the least interposition of new-formed connective tissue, and in nerve union it is of special importance.

Protection for United Nerves.—The ends of the nerves which are either in perfect apposition or face each other from a distance may

be protected from the invasion of connective tissue from the side by sterilized tubes. Decalcified bones were used by Vanlair. Payr used tubes of magnesium. Lotheisen prefers to protect the nerves with tubes of gelatin previously hardened in a two per cent solution of formalin. The gelatin tubes, after being treated by this solution, are preserved in 96 per cent alcohol; the lumen should be from 4 to 9 mm. Gelatin tubes can be sterilized for two hours by dry heat of from 110 degrees to 120 degrees. It has been my practice to protect the nerves from invasion of connective tissue by covering the point of suture with fascia, muscle substance, or fatty tissue. In the wrist or ankle areas, where muscle and fat are not available, gelatin and decalcified bone are good materials. The blood vessels of animals, always at hand, are good for this purpose. A mass made of equal parts of oil of sesame and paraffin, with a melting point of 107 degrees is especially good. This can be flattened out with a spatula almost as thin as paper and wrapped around the point of union, completely sealing the ends so that the neuroglia can supply the only immediate agglutinative material, which is readily penetrated by the axones.

Foramitti protected the points of anastomosis by surrounding them with arteries of the calf. The union is always perfect, and the point of anastomosis entirely free from the connective tissue in the vicinity. There is an insignificant adhesion between the intima of the artery and the sheath of the nerve. The arteries are prepared for this purpose in the following manner: A segment of artery from a calf is placed on a glass rod and hardened in 5 to 10 per cent of formalin solution for 48 hours, then it is washed in water for 24 hours, afterwards boiled for 20 minutes in water, and finally preserved in 95 per cent alcohol.

An ever-available material for this purpose is the membrane lining the inner surface of an egg-shell. The Cargile membrane may be used for the same purpose, as may also the peritoneum of a rabbit. Recently Murphy conceived the idea of wrapping around the point of anastomosis a roll of paraffin tape or a mixture of paraffin and wax, or simply a wax tape; however, these foreign materials were never used when a muscle or fascia could be obtained, either by flapping or splitting the muscle and inserting it where a normal muscle belly is directly available. The use of inorganic material is always somewhat hazardous.

THE SURGICAL ANATOMY OF THE FIELD OF OPERATION.

(Fig. 6.)

1. *Facial-Hypoglossal Operation.*—After the skin and superficial fascia and platysma are retracted, a number of small veins are encountered, branches of the anterior and posterior facial, external and internal jugular. The anterior border of the mastoid process is located, and the sterno-mastoid muscle. Then the parotid gland is exposed with the ramus and angle of the lower jaw. On separating the sterno-cleido-mastoid muscle backward, one comes to the posterior belly of the digastric, which, when drawn under the lower jaw, will expose the submaxillary gland. Now the common sheath of the internal jugular vein is seen with the internal and external carotid arteries below and anterior to it. On slight retraction backwards of the sterno-cleido-mastoid muscle and internal jugular, one can see shining through a loose areolar tissue, a whitish band, about one-eighth of an inch in diameter, which, when laid bare, is the hypoglossal nerve. This can be followed under the posterior belly of the digastric. At this point it lies on the internal carotid and some of the branches of the external carotid, as it comes down and curves forward, particularly the occipital artery.

The parotid region will show on its posterior border, the facial nerve entering and passing transversely under the auditory meatus to the anterior border, and under surface of the mastoid process. It lies at this point on top of the stylo-hyoid muscle, consequently the styloid process, which can always be felt. The transverse process of the atlas is situated at this point, and is the landmark or location of the beginning spinal accessory nerve.

2. *Facial-Spinal Accessory Operation.*—At first, the same structures come to view, but in order to obtain the view of the spinal accessory the internal jugular is drawn forward or towards the larynx, and the sterno-mastoid muscle is raised, when the upper branch, or the one supplying the latter muscle, is seen, and the greater branch passes down to the trapezius. (Shoulder.)

TECHNIC OF THE FACIAL-HYPOGLOSSAL ANASTOMOSIS.

After the usual local as well as general preparation the patient is placed under a general anesthetic.

Instruments.—Two knives, one regular size and one very small; one dozen fine artery forceps; two pairs of tissue forceps; two pairs of anatomic forceps; one Kocher director; one simple grooved director; two small dull retractors; two larger dull retractors; one

aneurysm hook; two pairs scissors, one straight, one curved; one pair of fine-pointed conjunctival forceps; one special facial nerve dissecting instrument (Fig. 7); one-half dozen fine small needles (round); one needle-holder.

Material.—Sutures, finest of silk, catgut, silkworm gut, decalcified chicken bone tubes, Cargile membrane, paraffin tape.

An incision is made, beginning close back of the auricle, midway between its upper and lower margins, carried downward and slightly forward in the direction of the hyoid bone. The length of the incision should be about four inches, and carried at first through the skin, superficial fascia and platysma muscle. These cut margins should be dissected loose and retracted. The external jugular vein, with its anterior and posterior facial veins, is exposed, ligated above and below, and severed. The deep fascia is carefully incised, preserving any of the branches of the superficialis coli nerves; the anterior border of the sterno-cleido muscle is exposed and followed upwards to the tip of the mastoid process. The posterior belly of the digastric is next exposed and the posterior border of the parotid gland by forcibly retracting the auricle and the skin over the ramus of the lower jaw forward. The anterior border of the mastoid process is followed along towards the tip into the depth, so as to reach the deeper layer of the parotid gland and to look for the entrance of the facial nerve into the latter, about on the level of the lower border of the external auditory meatus. The greatest mistake is made in looking for the facial nerve too near the surface and too far down from the ear. (One often finds here a superficial cervical branch.) After finding the facial nerve, which is recognized as a whitish cord, about the size of a Eustachian catheter No. 2, it is hooked up by means of the aneurysmal hook, and followed backward and upwards towards the stylo-mastoid foramen. This can best be located by following back the posterior belly of the digastric and the stylo-hyoid muscle immediately under it. The highest point of these two muscles will locate the foramen and the exit of the facial nerve. (Fig. 8.) In order to follow the nerve as high up into the foramen as possible, I have adopted and modified the ring instrument as devised by Mayo for dissecting varicose veins subcutaneously, and have added to it its ability to grasp. It is used in the following manner: Slip the ring over the nerve and follow it down to the foramen as high as possible and pull it out of canal. Place a sponge soaked in warm salt solution about it

and allow it to remain in the field. Next the hypoglossal is located by retracting the posterior belly of the digastric upwards under the lower jaw and the sterno-cleido-mastoid backwards. The common sheath of the vessels is exposed. This, with the sterno-cleido-mastoid muscle, is bluntly retracted. By bluntly dissecting some of the loose areolar tissue over the internal and external carotids, which can be felt pulsating with the finger, one can see a whitish structure passing over the external carotid just where the occipital artery is given off. This is the hypoglossal nerve which can be traced at this turn under the internal jugular and internal carotid upwards for a short distance, where it is lost sight of in its direction towards its foramen vertically into the cranial cavity. The distal course of the hypoglossal is forwards towards the tongue. At this point the technic will differ, whether we do an end to end or end to side anastomosis.

End to End.—(Fig. 9.) Follow the hypoglossal in its forward course to the tongue. The same ring-formed scissors may be used as for the facial. The nerve should be severed at the point, so as easily to meet the facial stump. The next step is to bring the cut ends into perfect apposition and suture them. One can use any of the methods described in the suture of nerves. The anatomosed nerves are now carefully placed into a pocket made within one of the muscles, preferably the posterior belly of the digastric, and a few superficial catgut sutures inserted, closing this pocket. All the structures of the neck are carefully replaced in their anatomic positions and wound closed without drainage, unless pus has been encountered, as in a suppurating gland, etc.

End to Side.—(Fig. 10.) Bring down the amputated facial nerve stump and find the location on the hypoglossal where the anastomosis will be best, that is, without producing any tension on either the facial or hypoglossal nerves. Having found that point, an incision, one-quarter inch long, is made with the small knife into the nerve sheath parallel with the course of the nerve. With the same knife about one-half of the axis-cylinders are severed within the sheath, that is, transversely, in order that when the facial stump is later implanted there will be direct axone contact, and thereby proper union, and still some of the fibers be left intact to perform part of the hypoglossal function. The next procedure is the preparation of the facial stump. The nerve sheath should be slit for about one-eighth of an inch and retracted so as to expose the

stump, that is, its axis-cylinders. The sutures are placed within it, but only through the sheath. Any method may be selected for suture of the nerves utilizing the slit. Fine conjunctival forceps are used by the assistant to separate this slit by allowing them to spring open. After the sutures are tied, these anastomosed nerves (Fig. 11) are placed within a pocket in a muscle (posterior belly of digastric) and treated in the manner already indicated. The wound is closed in the same way as in the other procedures.

TECHNIC OF THE FACIAL-SPINAL ACCESSORY ANASTOMOSIS.

The first steps of the preparation and operation are the same, except that the skin incision is not made so far curved forward, because the spinal accessory nerve lies more posterior than the hypoglossal. Locate the transverse process of the atlas; by well retracting the sterno-cleido-mastoid at the mastoid tip, one can hook up the spinal accessory nerve. If it is decided to use the trapezius branch, then that is dissected and severed, brought upwards and united to the facial. The anastomosed nerves are then placed within a pocket formed within the sterno-cleido-mastoid, or the posterior belly of the digastric muscle, and secured with two catgut sutures to prevent cicatricial formation at the new-formed union of the nerves, thereby preventing bad results.

POST-OPERATIVE OBSERVATIONS AND TREATMENT.

There are considerable soreness and pain in the regions operated on, and patients complain of some difficulty in swallowing and speaking for the first day or two. This is especially so if the hypoglossal is chosen. If closely questioned, most of the patients will state positively that they feel a sort of a tone in the paralyzed side of the face, but this soon disappears, and does not return until later, when gradual regeneration has taken place. Patients should be kept absolutely at rest for about a week, and as little motion of the head as possible should be made in order to insure good and rapid union of the nerve ends. The diet may be regular after the first forty-eight hours, if the patient is able to swallow well. The stitches may be removed at the end of a week or ten days.

Electric treatment should be begun as soon as the patient is walking about, in order to keep up the strength of the muscles and to stimulate the nerves. Mild faradic and galvanic currents are used as before operation. In case the hypoglossal is used, about the first signs that one observes of return of function are a sort

of contraction of the angle of the mouth, especially during the act of swallowing, and this may be observed as early as three months after operation. Gradually, the upper lip, and, finally, the eyelids, take on a toned-up and straight condition, contracting when the patient swallows. The patient declares very early after the operation that the food does not lodge within the cheek, and mastication on that side is so much more satisfactory. All other symptoms, such as lacrimation, etc., disappear. The test to reaction to degeneration shows return to the normal, that is, as described before. In case many nerve fibers of the hypoglossal have been cut, or all of them, there will be a tongue paralysis, which, outside of some speech difficulty, causes no untoward symptoms.

In case the spinal accessory nerve is used, then there will be the movements of the face associated with the movements of the shoulders, unless the whole trunk is used; then, of course, there will be a shoulder droop, a condition not very desirable, and perhaps worse than the facial paralysis. Otherwise, the same course of treatment is carried out as in the hypoglossal, and the same observations are made in reference to associated function.

REASONS FOR PREFERENCES AS TO NERVES SELECTED, AND METHODS.

An end to side anastomosis is preferable to any other method because the function of the tongue is not entirely destroyed and the desired cosmetic result on the face is obtained. The cortical, as well as nuclear, centers of the facial and hypoglossal nerves are much closer to one another, and it is believed that the function of the one is more easily transferred to the other than is the case with the spinal accessory. It is believed that the associated movements between the face and tongue on swallowing are more likely to be again disassociated. Again, the act of swallowing can better be contracted than the movement of the arm; it is very embarrassing to the patient to have his face move every time the arm is moved.

It is claimed that the end to end method is preferred because more axones come in direct contact with the opposite nerve, hore rapidly unite and regenerate. The spinal branch is used in preference to the hypoglossal because it is easily located and on account of its close proximity to the exit of the facial.

As to the use of the glossopharyngeal for anastomosis with the facial, I have been able to find only one reference.

REPORT OF CASES.

Case I.—Miss H., 42 years old. Has had a suppuration of her right middle ear since childhood, when she probably had Bezold's mastoiditis, as evidenced by a scar on her neck from multiple incisions. She came to me first in June, 1905, complaining very bitterly of pains in her ear and right sided head pains, foul smelling discharge, and a crooked face which she said had been existing only for a few months. She had been coughing for some time and generally feeling bad, losing flesh, and having night sweats. One of the principal complaints was a dizziness and a tendency to fall at times toward the right side. There was nothing important that could be elicited from the family history.

Examination.—A very frail, ill-nourished woman, weighing about 75 pounds. Temperature 99 1-2° (P. M.); pulse fair quality, and about 100. Nasal and throat findings negative. Right ear—external canal, negative, containing a fair amount of thick pus of a fetid character. After thoroughly removing it a practically normal tympanic membrane was observed, except in the lower posterior margin, where there was a small fistulous opening filled with granulations. Externally in the occipital regions marked tenderness was elicited by deep percussion on pressure. Test of hearing showed practically normal hearing through the whole range of Bezold, Edelmann, a positive Rinne. The left ear was also discharging some, but the patient knew nothing about it. There was a small perforation in the lower anterior quadrant. The hearing here was somewhat reduced and a slight negative Rinne-Weber lateralized to the left. After observation and local treatment for a short time, I decided to operate inasmuch as the patient complained more of her pains; especially when she attempted to lie on the right side of her head, she could not stand the pressure over the occipital region.

Operation.—Under general anesthesia the usual steps in radical operation were followed. When the annulus was resected and the middle ear cavity was explored, one could detect a necrotic area in the lower posterior portion of the middle ear, and that portion corresponding to the anterior extremity of the floor of the antrum, or posterior semicircular canal and the facial ridge. This area was very carefully curetted of the granulations, but I could not determine whether the semicircular canal or the facial nerve was injured at this point. I then proceeded to expose the painful

area over the occipital region, and therefore made a tongue-shaped flap, with the base at the neck, or say an inverted U incision. Peeling this down, including the periosteum, we had a fearful venous hemorrhage from the region of the lateral sinus. This was controlled by compression, and on closer examination of the flap, we found a flake of bone, the outer table of the lateral sinus. This bone, however, appeared healthy. I then exposed the sinus farther back toward the torcular herophili and by compression controlled the bleeding point. On closer observation I found some granulations about this lateral sinus and a fistula. By passing a flexible blunt pointed probe into the fistula it came out in the middle ear at the point spoken of above, namely, the lower posterior portion. Leaving this probe *in situ* I chiseled down upon it in order to transform it into an open wound. I firmly believe that the facial nerve was destroyed by the diseased process of the bone, or in any event I would very likely have injured it beyond the chance of spontaneous recovery, in order to eradicate the diseased process. The usual steps were then taken in finishing the plastic, having previously relieved the compression from the lateral sinus and tamponed the bleeding point of it. A retroauricular opening was left, in order to remove that tampon. The patient made an uneventful recovery and was perfectly free from pains and dizziness, but retained her facial paralysis. The reaction of degeneration showed plus, that is to say, no reaction resulted from the faradic current, when either applied to the nerve trunks, or muscles of the face, but there was a slow contraction of the muscles when the galvanic current was put on. That showed that the muscles were there and if union between the facial and another motor nerve took place, we could hope for results.

The photograph, Fig. 12, shows the condition and appearance before operation.

In the spring of 1906 the operation for facial-hypoglossal anastomosis was performed. Dr. Goldstein, St. Louis, Dr. Fleming, Los Angeles, and Dr. Owens, San Diego, California, were present and assisted. Reported in *Laryngoscope*, 1907. The operation as described in the previous chapter was performed and patient made an uneventful recovery.

Observation.—There was some soreness in swallowing and speaking which disappeared after two days. Patient declared voluntarily that there was a feeling in the right side of her face more like

the other side than before operation, but this disappeared in a few days. I am inclined to believe now that that was from the reaction of inflammation in the tissues about the neck. No visible change could be noticed for about ten weeks, when a sort of a contraction of the right side of the face was noted and when patient was asked to swallow, this contraction was more apparent. From this time on, this phenomenon became more clear and gradually this tonicity was noticed over a greater portion of the face until about five months after operation; the eyelids, upper and lower, took on the tonic condition. At this time whenever the patient only thought of swallowing, the face would move, and I began to think that that would be very detrimental to the success of this operation, but the patient soon learned to disassociate these movements and at the end of the year she could swallow by very slightly moving the face. At this time the test of reaction to degeneration was practically that as is found in normal cases. There was, however, this fact shown, that whenever patient laughed or smiled the healthy side of her face would respond, whereas the paralyzed side would lag and patient would have the characteristic appearance of facial paralysis. (Fig. 13.) There never was a voluntary movement of the facial muscles without the act of swallowing. The photograph, Fig. 14, which was taken about a year after operation, shows very well the patient as recovered so far as the cosmetic purposes go.

About this time the patient's cough again became generally worse and she decided to go to the country to be out in the fresh air. She wrote to me several times that she had lost her cough and was getting along well. In December, 1907, I wrote to her to come to Chicago as I wished to present her in connection with another case, and she came. When I examined her at this time, I found her in good health and her face straight. Two days later I was notified that she was taken to the hospital with acute lobar pneumonia, under the care of Dr. J. B. Herrick.

On the 10th day of the disease the patient died. The following day I made the post-mortem examination with these findings: General emaciation of the body. The face appeared symmetrical; field of operation of ear and anastomosis showed nothing different from antemortem. Calvarium section and removal of brain. No gross pathologic changes of the brain, venous sinuses, meninges, or bony structures in the vicinity of the temporal bones within the cranial

fossae. Neck dissection of nerve anastomosis. Tracing the anastomotic union of facial hypoglossal into the substance of the posterior belly of the digastric and liberating it, I found perfectly firm union of these two nerves. Examination of the right thoracic cavity showed the whole lung solid and old scars and some recent tubercular foci which were excised for histologic examination. There was some turbid fluid in the pleural cavity. Pleuritic adhesion in the postapical region. Left thoracic cavity showed some recent consolidation near the base, and an old healed tubercular apex. Heart apparently normal. Intra-abdominal viscera negative.

Histologic examination of lung structure showed characteristic changes of acute lobar pneumonia, and characteristic tubercular lung disease of the apex regions.

I then excised *lege artis* the wedge of the temporal bone including the structures of the neck which demonstrated the union of the facial-hypoglossal anastomosis and herewith present it, after carefully dissecting and preparing it, with the following findings:

The cavity of the middle ear, attic, aditus ad antrum and antrum are perfectly healed and epidermized. The posterior semicircular canal is plainly seen where it was exposed and opened, and now is healed. The region of the ostium tubae tympanicae is firmly closed off from the pharyngeal region. The resected (mastoid process) fistula from the region of the sigmoid sinus to the posterior inferior portion of the middle ear, as described in the operation, is found entirely healed. Slitting the sigmoid sinus along its course from the torcular herophili to the jugular bulb, I found it absolutely normal. The anastomosis is seen between the colored silk threads for demonstration purposes, namely, the centripetal end of the hypoglossal has a blue thread, the centrifugal end of the hypoglossal has red thread and the facial stump entering practically at right angles has a yellow thread tied about it.

Dissection of the stylo-mastoid region shows the facial nerve with yellow thread tied through it, demonstrating how very difficult it was to reach during the operation, a fact that caused me to suggest this instrument that can bluntly reach up to the stylo-mastoid foramen.

Of course, a histologic examination of the anastomosed nerves, as well as a more minute examination of the temporal bone, will follow and be of interest. This was impossible to do in order to demonstrate the gross specimen first.

Case II.—Mrs. M. This case has already been reported in the *Laryngoscope* in extenso, on account of the interesting pathologic condition of the temporal bone, so I will just briefly recapitulate in order to make clear the indication for the anastomosis.

She had an endothelioma intravascularis of the middle ear, which destroyed the continuity of structure of the facial nerve, with complete facial paralysis as a consequence. The radical removal of this tumor was not followed by any improvement of this paralysis, but there has been no recurrence of the growth for three years, an interesting fact since these growths are supposed to be sarcomatous.

The patient hesitated for over a year and a half before submitting to the nerve anastomosis. During this time this patient suffered from a general malnutrition and marked anemia which resisted all treatment. In 1907 she decided to have the operation performed, and a test of reaction of degeneration to the facial muscles was plus and a very weak response to the galvanic current, which showed that the muscles were very weak, and if union of the nerves did take place recovery would likely be very slow.

In 1907 the facial hypoglossal operation was performed, Dr Frank Allport and Dr. Otto Stein, Chicago, assisting. Great difficulty was encountered in finding the facial nerve inasmuch as there had been many scars in the mastoid region, especially low down anteriorly from a very long after-treatment of her first operation. When the facial was finally joined to the hypoglossal, end to side, there was considerable traction in the latter nerve. Again, imbedding this union into the posterior belly of the digastric and closing up the wound. A small granuloma was curetted from the external canal of the left ear, and miscroscopically proved not to be endotheliomatous.

Patient made an uneventful recovery from the operation with primary union of the skin incision. This patient made the same remarks, being a more intelligent patient than patient No. 1. She stated that her left cheek felt more natural and stronger. This also disappeared in the course of a week or two. After the patient came out of the anesthetic, I found the anterior and lateral portions of the left side of her tongue paralyzed, none of which was evidenced in Case 1. During the first six months following the operation, during which time the patient received daily electric massage and tonic treatments, there was very little improvement seen; in fact, so far as appearances go, only a beginning crease,

such as one sees as a natural contracture following the paralyzed muscles is observed, and only to be differentiated when the patient swallowed. From this time on the improvement was more in the upper branches; she could close her eyes better, and on the application of the faradic current, small contractions followed. However, I want to put this case on record up to the present day, not as a cure, but to prove the point that when one has a positive test of reaction to degeneration, and clinically has strong evidences that the nerve is very likely severed for a greater distance and scars, tumors, sequestra, or other foreign bodies, interposed between its ends, one should do the anastomosis as quickly as possible and not wait for nature to bridge it over. Another point is the general condition of this patient, namely, the anemia.

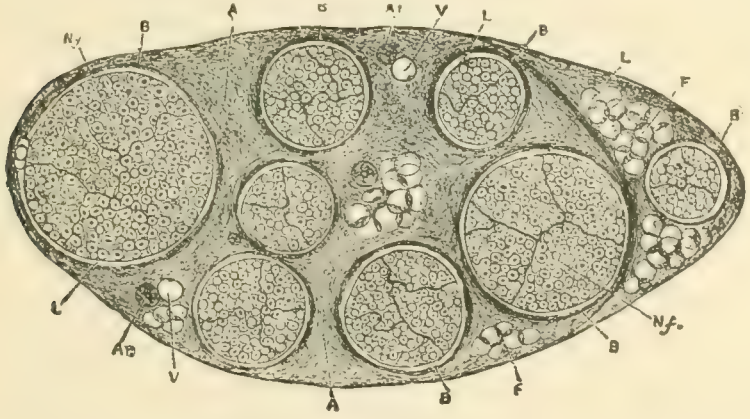

Fig. 2.—Transverse section of the sciatic nerve of a cat about x 100.—It consists of bundles (Funiculi) of nerve-fibres ensheathed in a fibrous supporting capsule, epineurium, A; each bundle has a special sheath (not sufficiently marked out from the epineurium in the figure) or perineurium B; the nerve-fibres Nf are separated from one another by endoneurium; L, lymph spaces; Ar, artery; V, vein; F, fat. Somewhat diagrammatic. (V. D. Harris.)

Case III.—Mr. M. This case has already been reported in connection with the report on Bezold's mastoiditis in *Journal of Opthalmology and Oto-Laryngology*, 1907, and I will therefore omit the detailed history. Suffice it to say at this time, that the man had a permanent facial paralysis associated with a marked necrosis of the temporal bone along the greater course of the facial nerve, including the nerve. On August 4, 1907, I made a facial-hypoglossal anastomosis, only that the technic differed in making it end to end. Drs. Ray, of Louisville, and Weaver, of Sherman, Texas, assisted.

There was again a great deal of difficulty in dissecting the facial, owing to many old scars on the neck from the Bezold mastoiditis many years before. We found quite a number of enlarged lymph glands, submaxillary, over the region of the hypoglossal that required dissection and took considerable extra time.

Observations.—After the patient came out of the anesthetic, he had complete paralysis of the left side of his tongue, and there was considerable difficulty in speaking and swallowing. This man had no such feeling of the paralyzed side of his face as in Cases I and II, after operation—that is, primary tonicity. Ten days after the operation, when the stitches were removed, there was found a small fistula in the region of the parotid gland, discharging saliva, and

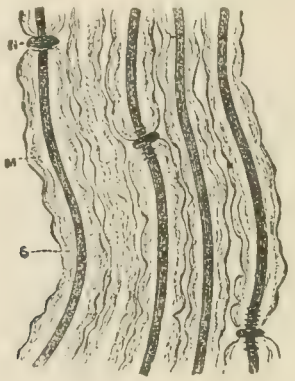

Fig 3.—Several fibres of a bundle of medullated nerve-fibres acted upon by silver nitrate to show peculiar behavior of nodes of Ranvier, N, toward this reagent. The silver has penetrated at the nodes, and has stained the axis-cylinder, M, for a short distance. S, the white substance. (Klein and Noble Smith.)

when the patient would suck a lemon, this flow would be markedly increased. This fistula was very likely produced during the manipulations to dissect the facial nerve by a sharp retractor. This precaution against such an accident is brought out by Murphy.

As to the course of this case, I am not able to report any further than that he left Chicago about one month after the operation, having had no after-treatment. Being a laborer and in very bad circumstances, he went to Cleveland to seek employment. About four months after the operation I received a letter from Dr. Large, who chanced to see him in his clinic, and he states that the face appears completely paralyzed, and the tongue paralysis is giving the

patient considerable trouble. No conclusion can be drawn from this report, however, as a test for reaction of degeneration, and the observations of one who has seen the case before operation are necessary.*

Case IV.—Mrs. S., 32 years old. Suffered for a long time with tic douloureux and facial paralysis on the left side. When I first saw her she had received every possible treatment for the cure of her tic, including the injection of osmic acid (Murphy); neurexere-

Fig. 4.

sis of the three branches and resection of the Gasserian ganglion (by Carl Beck) without positive relief. Examination of the ear showed no evidences of trouble there, and the test of reaction to degeneration was negative. The eyelids and forehead muscles were not affected. The eye could not rotate outwardly, and there was a paralytic squint. She could not hear in this ear. Therefore, I made a diagnosis of some central pathologic lesion involving

the fifth, sixth, seventh and eighth nerves, probably tubercular, as there had been a history of this affection.

On January 7th, 1907, I operated, reopening the original bone flap that had been made by Dr. Carl Beck in operating for the Gasserian ganglion, and found a very markedly thickened dura. (Sectioned later and found just granulations, extradural, but no tuberculosis.) Elevating the brain structure I found considerable matting together of the base of the brain through the dura. I ex-

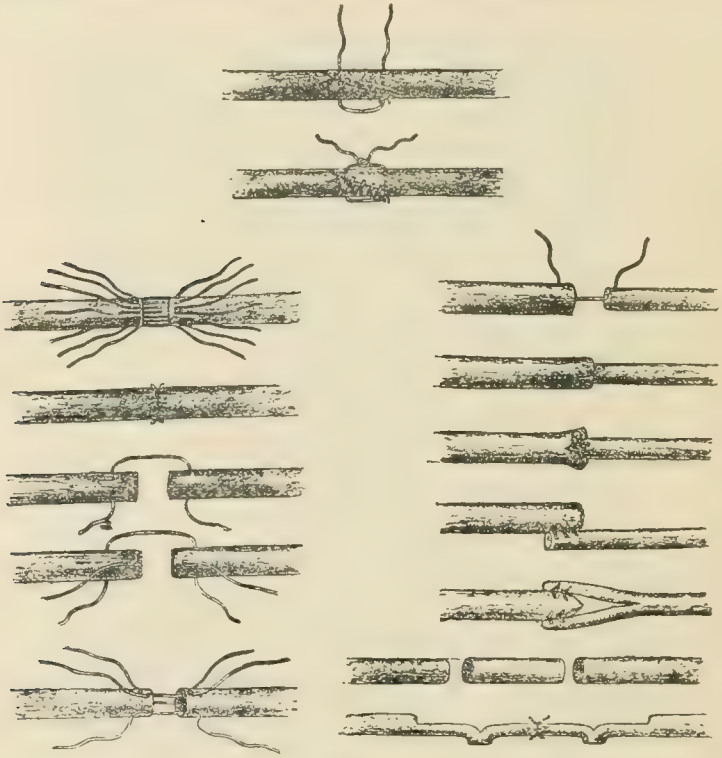

Fig. 5.

plored the region of the exit of the above nerves from the brain and did not find any gross lesion. Knowing that exploring this region of the brain structure would very likely kill the patient, I stopped the operation. However, before closing up I explored the brain from above in various directions and found nothing in the shape of a tumor. To my astonishment the patient recovered and was free from the neuralgic pains. Enthusiasm on my part and the desire of the patient to have her face straightened, I agreed to do a facial

hypoglossal anastomosis. The hypoglossal nerve was of course not involved in the above intracranial trouble. On February 5, 1907, I operated in the usual way, intending to do the end to side procedure. Dr. Vaughan, Chicago, and Dr. Edwards, of Kansas, assisted. When I had the facial dissected and excised from the stylomastoid foramen and attempted to bring it into close proximity to unite with the hypoglossal, I found it impossible. Having just

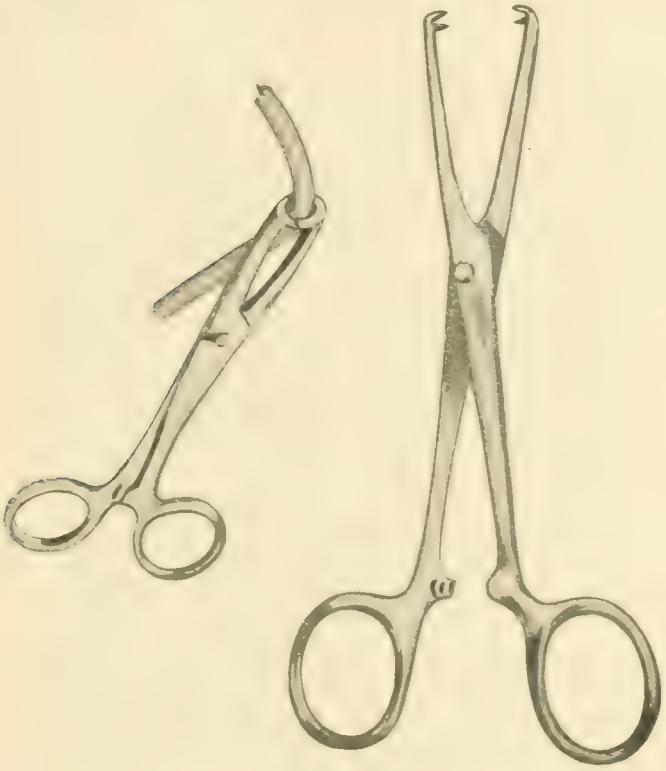

Fig. 7.

read of Gluck's experiments to use living tissues as conductors for nerve fibers to regenerate, I used a neighboring large branch of the superficial cervical nerves. Excising a long enough piece, I made with it an end to end procedure with the facial, and implanted it into a slit of the hypoglossal as usual. This whole area was covered by a layer of the sterno-cleido-mastoid muscle, that being nearest at hand, to prevent adhesions. Patient recovered from this operation without any untoward symptoms. Shortly after, however,

nephritis was diagnosed and a general anasarca followed. The patient did not live more than two months after the performance of this last operation, and consequently I can add nothing further to the subject. There was never any statement made by the patient that she felt any primary tone on the operated side of the face, and no evidences of recovery were demonstrable. The patient had some trouble in swallowing and the tongue lagged considerably on the left side.

Inasmuch as a post-mortem was not permitted, I cannot say what the conditions of the anastomotic points were, nor can I possibly find out the specific etiologic factor.

Case V.—Mr. W. B. D., Louisville, Ky., aged 53 years. Has always enjoyed good health until he developed an earache in right

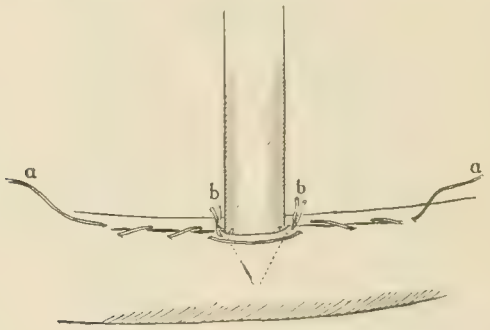

Fig. 11.

ear, which was diagnosed as an abscess by Dr. Cheatam, who lanced it and applied leeches, but without relief. Pains did not diminish. He was taken to infirmary after two days' time and drum again incised. Hearing was never much interfered with and there was never much soreness back of ear. Deep-seated pain over right eye. Expectant treatment was instituted, consisting of repeated irrigation of the ear with hot water and lying on hot water bag; this was continued for six weeks, when treatment was changed to flaxseed poultice every 20 minutes for about 72 hours. After elapse of one day Dr. Cheatam performed mastoid operation, following which there was no relief from pain. Six days later second operation was performed by Dr. Cheatam, assisted by Dr. Ray, following which the patient still was not relieved from pain and eye

aching. Immediately after recovery from anesthesia facial paralysis was observed.

Findings at time of operation given by Dr. Cheatam and Dr. Ray are as follows:

These deep-seated head and eye pains continued for about three months, gradually abating until they disappeared. The healing of wound back of ear required a considerable length of time, about five months. The discharge from the middle ear ceased a week

Fig. 12.

or ten days after, never recurring. The facial paralysis had never taken on any change in spite of all local and general treatment. Besides difficulty in speech and cosmetic appearance there was a metallic taste in the anterior and lateral half of tongue (right side), which was constant, especially when he cleared his throat. Lacrymation considerable. He did not use right side of face for chewing, as the food lodged in the cheek cavity. Patient thought that sali-

vary secretion and taste might have diminished some. Olfaction did not seem to have been affected. Breathing (nasal) as good as ever. General health has been good except he had some trouble with bladder with bloody urine, which would clear up and return. There was some difficulty in urination, sometimes burning. Examination of urine was made by Dr. Wendell, Dr. Wathen, Jr., Dr. H. Koehler, and kidneys found normal, but there had been no general examination made since operation. Appetite good, patient sleeps well, bowels regular. Habits are, practically abstainer.

Examination.—Characteristic facial paralysis. There was primary contracture at lower lip extending to the margin of lower jaw. Lower lid drooped so as to evert lacrymal duct down and out, showing accumulation of tears constantly in lower sac. The alae of nose, right side drooping, narrowing nostril. Upper lip hung over margin of upper teeth, and lower lip, caused by upper lip, held closer to lip margin. At back of the ear was found evidence of an incision leading from a line drawn across the upper margin of auricle downwards to about an eighth of an inch from the tip to lobule, in middle of which there was a depression in the mastoid process of the size of a lima bean. In the upper margin of incision there was a keloid scar firmly adherent to bone.

Patient was observed last October under superficial examination, but a positive examination made as to a reaction degeneration, which herewith is repeated:

Test of Reaction of Degeneration.—No response to the faradic current. When galvanic current is applied patient feels contractions at make and break, but not observed by myself.

Nasal Examination.—Practically negative with a few irregularities.

Examination of Tongue.—Perfectly straight.

Examination of Palate.—Shows absolute movement normal, the muscle of uvula reacting promptly.

Examination of Right Ear.—Stenotic canal (externally). The tympanic membrane seems to be entirely regenerated. No evidence of disease in remaining portion of canal.

Examination of Hearing.—Hears highest tones. Hears low tuning fork. Weber laterates to right. Rinne negative. Shorter on right side. Rinne positive on left side.

General Examination.—Revealed no pathologic condition as to his kidneys, gastro-intestinal tract and heart and lungs. February

12, 1908, facial-hypoglossal anastomosis was performed, Dr. Ewing M. Day, Pittsburgh, Dr. J. Pratt, Shelbyville, Ky., Dr. L. J. Hughes, of Chicago, assisting. I found the facial nerve unusually easy in this case, for in the former cases this procedure lasted very long. On account of the long continued drainage after the mastoid operation, I expected much trouble from adhesions. We made the end to side implantation and buried the nerve in the pos-

Fig. 13.

terior belly of digastric. Time of operation 2 hours and 48 minutes.

Observations.—With the exception of a marked nausea, which he said he had also with his other operations, the patient recovered very well and continued to remain normal for five days, when his temperature rose to 103.4°. Examination of the wound showed a marked swelling in the region of the parotid and along the lower jaw. Releasing some of the upper sutures a flow of saliva of es-

tablished (salivary fistula), and on the following morning he had a temperature of 99°. Every day following until this day, February 21, 1908, his temperature fluctuated between 99° and 102°, with some degree of uncomfortable feeling. Examination of the pharynx shows a small degree of edema of the uvula. There can be no further statements made at this early time as to results.

CONCLUSIONS.

The fact that one case out of a dozen of facial-hypoglossal anastomosis will recover is sufficient encouragement and indication to justify its performance.

In order not to bring this procedure into disfavor, one should select his cases and this selection should be based upon a thorough examination, especially as to the reaction of degeneration. That is to say, the muscles must be present, no matter how flaccid, and the galvanic electric current will bring that out, as stated above.

A thorough knowledge of the anatomy and technic is absolutely necessary and I know of nothing better to recommend than practice suturing of nerves on dogs and perform the operation on the cadaver two or three times before doing the operation.

One should not be satisfied by simply doing the operation, but great care should be exercised in the long, continuous after-treatment of electricity, massage, etc.

Since the reading of this paper, the author has operated on the sixth case which has the following history:

Mr. B., 18 years of age, referred to me by Dr. Casey Wood, when 1½ years old had a marked mastoid disease with secondary abscesses in the neck, anterior and posterior, as well as anterior to the auricle in the parotid regions of the left side. Dr. Barkan, of San Francisco, operated at the time, and it required a great length of time before boy recovered. His face, previous to the mastoid operation, was straight. Following the procedure he had complete facial paralysis.

Examination.—Characteristic picture of the condition with all the usual symptoms. A number of large scars are found on his face, neck and back of the ear. In this locality is quite a large fistula, communicating with the auditory canal, but it is entirely dry. Test of reaction to degeneration shows it is negative, that is, there is no response to any kind of current, and consequently a case with very poor prospects of recovery. In fact, from the length of time

that the paralysis has existed one would not expect any result, and is a contraindication to the operation.

I wish, however, to state, that as this is the son of well-to-do parents, who took advantage of everything known to them to aid the boy in recovering function in the paralyzed side of his face, the patient has, all these years had stimulating treatments to the nerves and muscles of the face. Consequently there was some chance that remnants of muscles might be present.

Fig. 14.

Dr. Casey Wood and the father decided, nevertheless, to have me try to do an anastomosis and take a chance on the result.

Operation.—Through one of the existing neck scars I soon reached the region of the facial nerve. To my great disappointment I found the complete absence of any such structure and, if it was present at all, it was not recognizable as a facial nerve, nothing resembling a nerve structure in the whole course. I dissected

towards the face and here also found no trace of any of the branches. Consequently I closed up the wound and stated to the parents the absence of the facial nerve. The young man made an uneventful recovery from the operation.

Two weeks later I performed a plastic on his face in the following manner: Through a scar situated over the zygomatic region which was first resected, a long straight needle armed with strong silk was passed well under the skin towards the labial fold, there made a loop, and needle again made to come through the original entrance opening. Drawing the thread taut, somewhat puckering the skin so as to make an artificial labial fold, the threads are tied and the wound closed by primary intention. Patient recovered with a very good cosmetic result, as the accompanying pictures show.

Case IV.—Mr. M. has reported since the reading of the paper and sent a photograph. It shows distinctly a formation of labial fold and he reports associated movements of his face with the act of swallowing. The tone of the paralyzed side has much improved.

Case V.—Mr. D. Doctor Moren, of Louisville, reports that the muscles of his paralyzed side of his face are responding much better and patient declares an improvement in the tonicity of his face. z

Case VI.—Mrs. M. has an improvement in her facial expression but her general condition of anemia is constantly increasing and the chances of recovery are not very good.

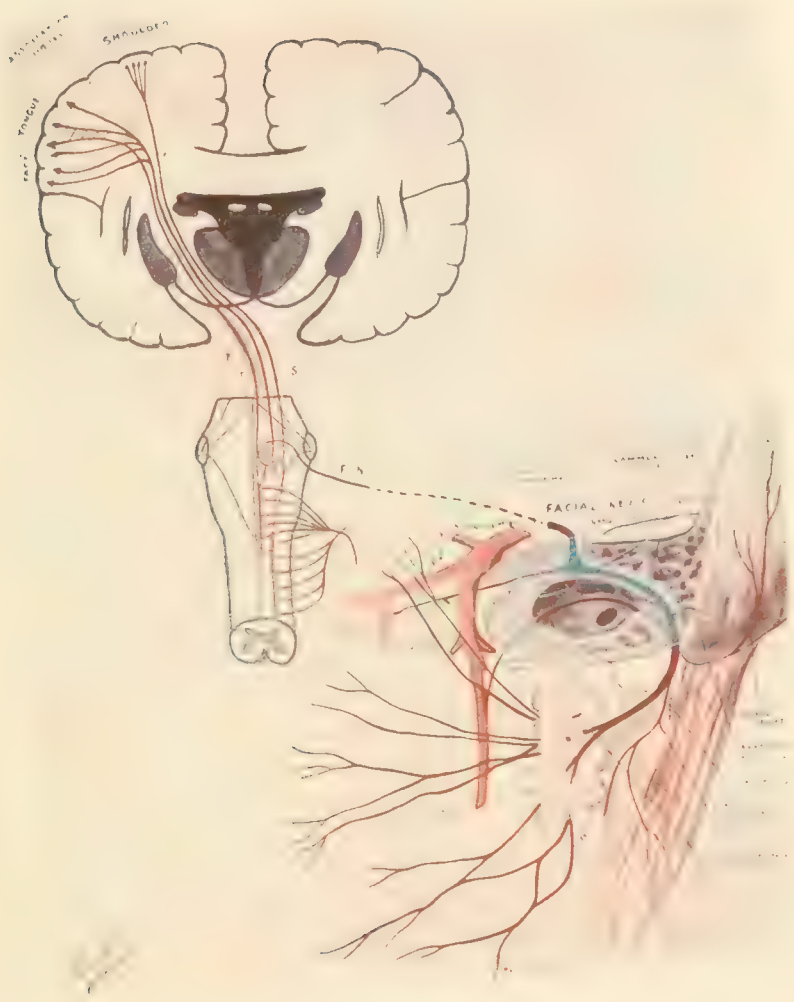

Fig. 1

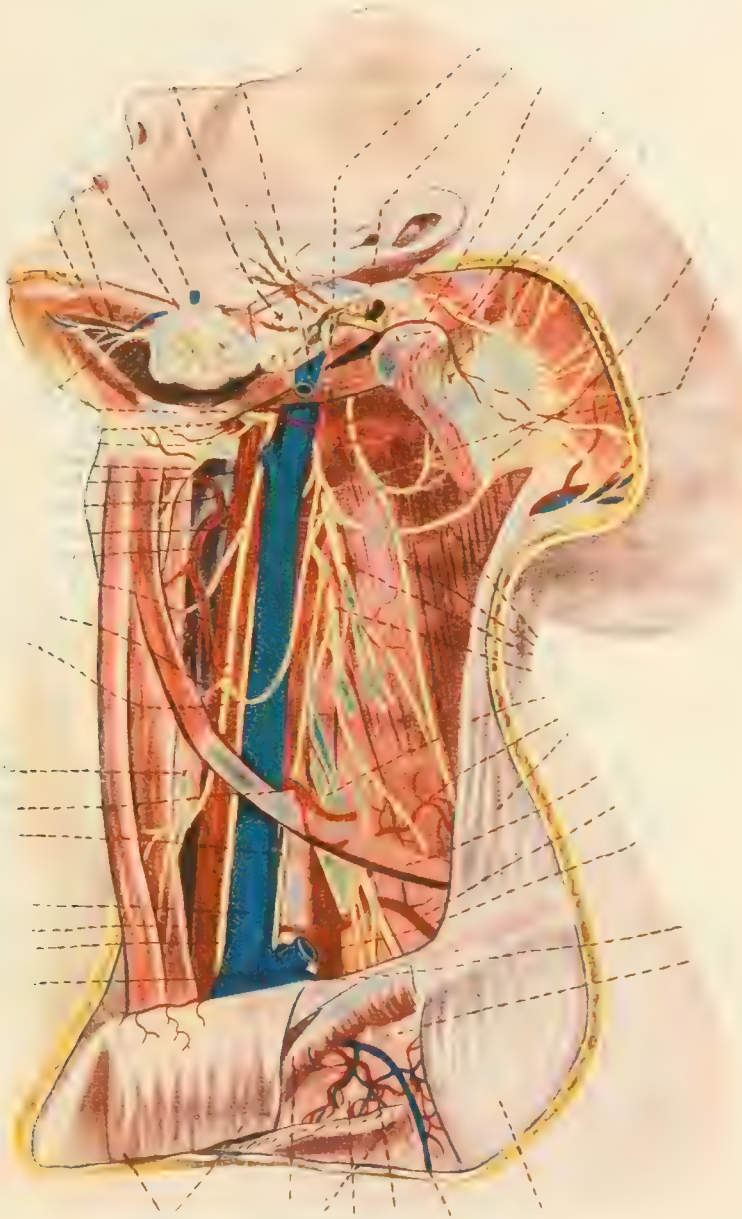

FIG. 6.

See next page

FIG. 6.

Fig. 6—Represents the surgical anatomy of the field of operation of the facial nerve with the other two nerves spinal accessory and hypoglansal .

1. Sternocleidomastoid artery.
2. Facial nerve.
3. Occipital branch of posterior auricular artery.
4. Posterior auricular branch of facial nerve.
5. Mastoid tip.
6. Sterno mastoid, muscle cut and retracted.
7. Lesser occipital nerve.
8. ii Cervical nerve (ventral Br).
9. Accessory nerve.
10. Accessory nerve.
11. iii Cervical nerve (ventral br).
12. iv Cervical nerve (ventral br).
13. Brachial plexus.
14. Ascending branch of transverse cervical.
15. Trapezius.
16. Omohyoidens.
17. Descending branch of transverse cervical artery.
18. Subclavian artery.
19. Subclavian vein.
20. Pectoralis minor.
21. Deltoidens.
22. Cephlic vein.
23. Thoracs acromial vessels.
24. Clavicular portion of pectoralis major.
25. Ant. Thoracic nerve.
26. Clavacular port. of pectoralis major.
27. Sternocleidomastoidens.
28. Sternocleidomastoidens.
29. Sternocleidomastoidens.
30. Innominate vein.
31. Vagus nerve.
32. Com. cartoid nerve.
33. Inf. bulb of int. jugular vein.
34. Phrenic nerve.
35. Super. cervical artery.
36. Thyroid glands.
37. Ansa Hypoglossi.
38. Descending br. of hypoglossi.
39. Ascending br. of cervical artery.
40. Superior thyroid artery.
41. Descending br. of hypoglossal.
42. Superior laryngeal artery.
43. Ext. caroted artery.
44. Thyreohyoid br. of hypogl.
45. Lingual artery.
46. Hypoglossal nerve.
47. Stylohyoidens.
48. Digastricus.
49. Submental artery.
50. Mylohyoid nerve.
51. Submaxillary gland.
52. Ant. facial vein.
53. Paroted gland.
54. Posterior facial vein.

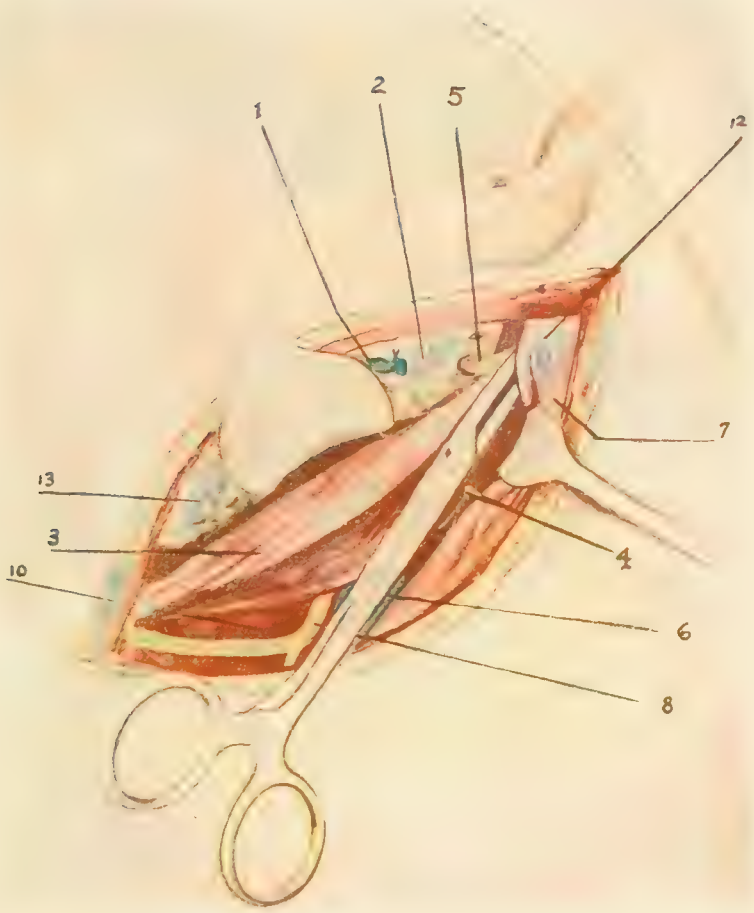

Fig. 8.

1. Ant. facisl vein.
6. Int. jugular vein.

FIG. 9.

Fig. 9. Representing the [illegible] method.

1. [illegible] vein.
12. Mastoid tip.
2. Parotid gland.
3. [illegible] muscle.
4. Post. belly of digastric M.
5. [illegible] down under stylo-hyoidens, [illegible] Nr. 11
6. Internal jugular vein.
7. Sternocleidomastoidens muscle.
8. Hypoglossal nerve.
9. Hypoglossal nerve, its descending branch.
11. Hypoglossal nerve with the facial stump implanted).
10. Common carotid [illegible] subdivisions.
13. Submaxillary gland.

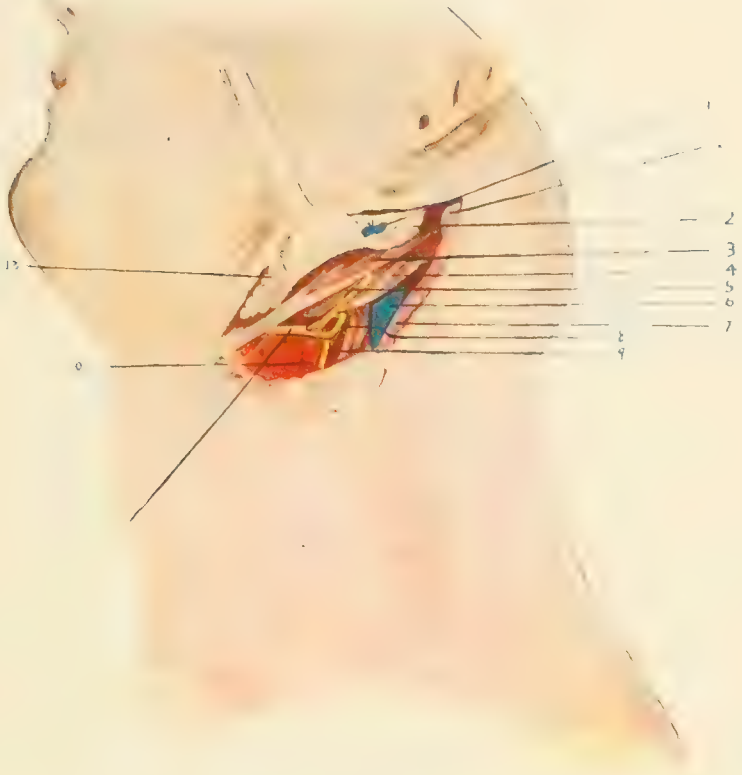

Fig. 10

Representing the facial hypoglossal anastomosis by the end to side method

1. Ant. facial vein.
2. Parotid gland.
3. Stylohyoideus.
4. Post. belly of digastricus.
5. Facial nerve cut and pulled under the stylohyoideus.
6. Internal jugular vein.
7. Sternocleidomastoideus.
8. Hypoglossal nerve.
9. Hypoglossal nerve, descending branch.
10. Common carotid artery.
11. Hypoglassal nerve.
12. Mastoid tip.
13. Submaxillary gland.

BUSINESS MEETING.

The fourteenth annual meeting of the American Laryngological, Rhinological and Otological Society convened at Pittsburg, Pa., May 28, 29 and 30, 1908.

The Society was called to order by the President, Dr. Ewing W. Day, of Pittsburg, at 10 A. M., May 28.

During the sessions of the Society the following Fellows and visitors registered:

Dr. Frank A. Allport, Chicago, Ill.
Dr. Willis S. Anderson, Detroit, Mich.
Dr. William L. Ballenger, Chicago, Ill.
Dr. William C. Bane, Denver, Colo.
Dr. John F. Barnhill, Indianapolis, Ind.
Dr. Joseph Beck, Chicago, Ill.
Dr. William S. Bryant, New York City, N. Y.
Dr. R. Bishop Canfield, Ann Arbor, Mich.
Dr. Lewis A. Coffin, New York City, N. Y.
Dr. Eugene A. Crockett, Boston, Mass.
Dr. William Ledlie Culbert, New York City, N. Y.
Dr. H. Holbrook Curtis, New York City, N. Y.
Dr. Ewing W. Day, Pittsburg, Pa.
Dr. Edward B. Dench, New York City, N. Y.
Dr. Arthur B. Duel, New York City, N. Y.
Dr. H. Bert Ellis, Los Angeles, Cal.
Dr. Thomas J. Gallaher, Denver, Colo.
Dr. Max A. Goldstein, St. Louis, Mo.
Dr. Charles Graef, New York City, N. Y.
Dr. Charles P. Grayson, Philadelphia, Pa.
Dr. Thomas H. Halsted, Syracuse, N. Y.
Dr. Thomas J. Harris, New York City, N. Y.
Dr. Henry J. Hartz, Detroit, Mich.
Dr. William H. Haskin, New York City, N. Y.
Dr. Christian R. Holmes, Cincinnati, Ohio.
Dr. Edgar M. Holmes, Boston, Mass.

Dr. Thomas Hubbard, Toledo, Ohio.
Dr. E. Fletcher Ingals, Chicago, Ill.
Dr. John M. Ingersoll, Cleveland, Ohio.
Dr. Chevalier Jackson, Pittsburg, Pa.
Dr. Clarence Porter Jones, Newport News, Va.
Dr. Samuel J. Kopetzky, New York City, N. Y.
Dr. F. H. Koyle, Hornellsville, N. Y.
Dr. D. A. Kuyk, Richmond, Va.
Dr. John J. Kyle, Indianapolis, Ind.
Dr. George A. Leland, Boston, Mass.
Dr. H. W. Loeb, St. Louis, Mo.
Dr. Stephen H. Lutz, Brooklyn, N. Y.
Dr. John S. Mabon, Allegheny, Pa.
Dr. G. Hudson Makuen, Philadelphia, Pa.
Dr. P. E. D. Malcolm, New York City, N. Y.
Dr. William Beverly Mason, Washington, D. C.
Dr. Charles S. Means, Columbus, Ohio.
Dr. Robert Milligan, Pittsburg, Pa.
Dr. John W. Murphy, Cincinnati, Ohio.
Dr. Robert C. Myles, New York City, N. Y.
Dr. James F. McCaw, Watertown, N. Y.
Dr. Samuel McCullagh, New York City, N. Y.
Dr. James F. McKernon, New York City, N. Y.
Dr. O. A. M. McKimmie, Washington, D. C.
Dr. Wendell C. Phillips, New York City, N. Y.
Dr. Norval H. Pierce, Chicago, Ill.
Dr. Kaspar Fischel, San Francisco, Cal.
Dr. John B. Rae, New York City, N. Y.
Dr. Henry O. Reik, Baltimore, Md.
Dr. W. Scott Renner, Buffalo, N. Y.
Dr. George L. Richards, Fall River, Mass.
Dr. Charles W. Richardson, Washington, D. C.
Dr. John O. Roe, Rochester, N. Y.
Dr. Dunbar Roy, Atlanta, Ga.
Dr. C. C. Sandles, Pittsburg, Pa.
Dr. B. R. Shurley, Detroit, Mich.
Dr. Harmon Smith, New York City, N. Y.
Dr. S. MacCuen Smith, Philadelphia, Pa.
Dr. Herbert E. Smyth, Bridgeport, Conn.
Dr. Amos R. Solenberger, Colorado Springs, Colo.

Dr. Frank B. Sprague, Providence, R. I.
Dr. Charles Nelson Spratt, Minneapolis, Minn.
Dr. J. A. Stucky, Lexington, Ky.
Dr. C. A. Thigpen, Montgomery, Ala.
Dr. John A. Thompson, Cincinnati, Ohio.
Dr. John J. Thomson, New York City, N. Y.
Dr. Derrick T. Vail, Cincinnati, Ohio.
Dr. Eugene L. Vansant, Philadelphia, Pa.
Dr. Henry L. Wagner, San Francisco, Cal.
Dr. Ernest DeW. Wales, Indianapolis, Ind.
Dr. Joseph A. White, Richmond, Va.
Dr. John R. Winslow, Baltimore, Md.

The President opened the meeting with an informal address.

The reports of the Sections were then presented by the respective Chairmen.

REPORTS OF SECTION MEETINGS.

Report of the Southern Section.

The Southern Section of the American Laryngological, Rhinological and Otological Society held its annual meeting in Baltimore, December the 27th and 28th, 1907, Dr. H. O. Reik acting as Chairman. The epidemic of grippe, which extended over the entire country kept away many who had signified their intention of attending the meeting, but the local profession, who had been especially invited to participate, together with the following named members of the society, constituted a fair audience. Among those present were: Drs. Harry L. Myers, Norfolk, Va.; H. S. Hedges, Charlottesville, Va.; W. A. Wells, Washington; W. B. Mason, Washington; J. F. Woodward, Norfolk, Va.; Emilio Martinez, Havana, Cuba; John F. Culp and J. W. Park, Harrisburg, Pa.; E. B. Dench and Thos. J. Harris, New York; G. H. Makuen and H. J. Off, Philadelphia; J. R. Winslow, H. O. Reik and Geo. Rueling, Baltimore.

The first session was called to order at 2:30 p. m. in the parlors of the Hotel Belvedere, and the following papers read:

1. A plea for conservatism in the treatment of certain affections of the nose and ear.—Dr. Harry L. Myers, Norfolk, Va.

2. An unrecognized foreign body in the larynx.—Dr. Emilio Martinez, Havana, Cuba.

3. Report of a remarkable case of sigmoid sinus thrombosis, with multiform septic complications extending over a period of 4½ months. Ultimate death.—Dr. John R. Winslow, Baltimore, Md.

4. Some interesting cases of lateral sinus thrombosis.—Dr. H. Friedenwald, Baltimore, Md.

The session of Saturday morning, December 28th, was held at the Johns Hopkins Hospital, where Dr. Harvey Cushing opened the proceedings with an operation in the amphitheater of the new surgical building, performing the operation of cerebral decompression after his own method.

Dr. W. S. Halsted followed with a most interesting discourse on a subject which was new to the members of the society and of the utmost importance to them, namely, "Incipient expansion of the carotid ventricle (aneurism of the carotid)—The symptoms referred to organs of special sense.—Treatment with the metal band.

Dr. J. M. T. Finney then gave his personal experiences with plastic operation to correct nasal deformities resulting from destruction of the bridge.

4. "The tonsillar ring and paranasal sinuses as portals of entry for infectious diseases," by Dr. Sylvan Rosenheim.

5. Exhibition of a case of pan-sinusitis," by Dr. J. F. Culp, Harrisburg, Pa.

The afternoon session of Saturday was convened at the Hotel Belvedere and opened with a paper by the secretary of the Society, Dr. Thomas J. Harris, New York, entitled—"The prognosis in progressive middle ear deafness."

2. "The relationship between degenerate tonsils and middle ear deafness."—Dr. J. N. Reik, Baltimore.

3. Membranous pharyngitis accompanying influenza."—Dr. J. F. Woodward, Norfolk, Va.

4. "The contour and contents of the epitympanum in relation to suppurative diseases of the middle ear."—Dr. C. J. Blake, Boston. (Read by the chairman.)

5. "The clinical treatment of intracranial diseases of otitic origin."—Dr. J. A. Stucky, Lexington, Ky. (Read by the chairman.)

6. Exhibition of new instruments.—Dr. F. R. Sprague, Providence.

H. O. Reik, Chairman.

Report of the Eastern Section.

The meeting of the Eastern Section of the American Laryngological, Rhinological and Otological Society, held in Boston, January 4, 1908.

The morning session was held at the Massachusetts Eye and Ear Infirmary, and at the Massachusetts General Hospital, Throat Out-Patient Department. It was entirely a clinical session, with demonstrations of various cases, particularly fourteen results of the Killian operation for frontal sinus disease and a case of sarcoma of the temporal bone treated by Coley's serum. At the Massachusetts General Hospital Dr. Chevalier Jackson demonstrated the use of his method of direct inspection of the stomach on four cases.

The members of the Section lunched together at the Harvard Medical School.

The afternoon session was held in one of the lecture rooms of the Harvard Medical School. Papers were read by Drs. Robert H. Craig, J. Payson Clark, Norton L. Wilson, William E. Chenery, D. Crosby Greene, Francis P. Emerson, George L. Richards, George L. Tobey, Jr., and W. Sohier Bryant.

In the evening the members of the Section dined together at the University Club.

The meeting was a profitable one, and the promised attendance was large, but at the last moment the prevailing epidemic of influenza raging through the East cut down the attendance at the meeting materially. At the last moment nine telegrams were received, stating that either the members were ill or that there was illness in the members' families. In spite of all this, an attendance of twenty-four was secured.

E. A. Crockett, Chairman.

Report of the Middle Section.

The meeting of the Middle Section of this Society was held at Indianapolis, Indiana, February 22d, 1908, under the chairmanship of Dr. John F. Barnhill, of Indianapolis, Ind.

There were present thirty members of the Society, including the President Dr. Ewing W. Day, and in addition about thirty visitors were in attendance.

The chairman welcomed the members of the Society to Indianapolis, after which there was a Symposium on the Suppurative Diseases of the Temporal Bone. Papers were read as follows:

1. The Causation, by Dr. Wm. E. Sauer, of St. Louis, Missouri.

2. The Pathology and Bacteriology, by Dr. Earnest De Wolfe Wales, of Indianapolis, Indiana.

3. The Treatment Other than Surgical, by Dr. George F. Keiper, of Lafayette, Indiana.

4. The Heath Operation, by William L. Ballenger, of Chicago, Illinois.

5. The Radical Mastoid Operation, by Dr. John J. Kyle, of Indianapolis, Indiana.

The Symposium was discussed by Drs. O. J. Stein, Chicago; William L. Ballenger, Chicago; J. A. Stucky, Lexington, Ky.; R. Bishop Canfield, of Ann Arbor, Mich.; Joseph C. Beck, Chicago, Ill.; Derrick T. Vail, of Cincinnati, O.; George F. Keiper, of Lafayette, Ind.; William E. Sauer, St. Louis, Mo.; M. A. Goldstein, St. Louis, Mo.; Ewing W. Day, Pittsburg, Pa.; Frank Allport, Chicago, Ill.; John F. Barnhill, Indianapolis, Ind.; Hanau W. Loeb, St. Louis, Mo.; John A. Thompson, Cincinnati, O.; and the discussion closed by Drs. Keiper, Ballenger and Kyle.

On motion, the Section adjourned until 2:30 p. m.

AFTERNOON SESSION.

The Section reassembled at 2:30 p. m., and was called to order by the Chairman.

Dr. L. Page, of Lafayette, Ind., read a report of two cases of carcinoma of the larynx. He also reported a case of tuberculosis of the gum and lip.

Dr. Daniel W. Layman, of Indianapolis, Ind., reported and presented a case in which deficient cerebration was a prominent symptom in otitis media purulenta, which was cured by a radical mastoid operation.

Dr. John F. Barnhill reported two cases of brain abscess with recovery.

The cases of Drs. Layman and Barnhill were discussed by Drs. M. A. Goldstein, Joseph C. Beck, R. Bishop Canfield.

Dr. L. Page exhibited a case of suppuration of the labyrinth, which was operated on two years ago.

Dr. M. A. Goldstein presented a specimen of cyst of the tonsil.

Dr. Joseph C. Beck reported cases and exhibited new instruments in connection with treatment by opsonic method. Dr. Beck's report was discussed by Dr. Frank Allport.

Dr. Frank Allport, of Chicago, read a paper entitled "The Operation for Sinus Infection."

Dr. William R. Dabney, of Marietta, Ohio, read a paper in which he reported several cases of operation for sigmoid sinus infection.

Dr. J. A. Stucky, of Lexington, Ky., made some remarks on "The Operation for Brain Abscess of Otitic Origin."

Dr. R. Bishop Canfield, of Ann Arbor, Mich., read a paper entitled "The Operation for Labyrinth Suppuration."

Dr .Joseph C. Beck, of Chicago, Ill., followed with a paper on "The Surgery of the Facial Nerve."

On motion, the Section then adjourned *sine die*.

J. F. Barnhill, Chairman.

Report of the Western Section.

The Western Section of the American Laryngological, Rhinological and Otological Society met at Hotel del Coronado, April 21st, 1908, in conjunction with the California State Medical Society. Our Section took charge of the Ear, Nose and Throat program for the State Society. Some fifty physicians were in attendance. The program was as follows:

1. Some Practical Points in the Diagnosis and Treatment of Tuberculosis of the Ear and Upper Air Passages—Dr. F. M. Pottenger, Los Angeles.—by invitation.

2. Acute Otitis Media—In Infants and Children. Dr. H. Bert Ellis, Los Angeles.

3. Acute Otitis Media—Prophylaxis and Treatment.—Dr. William Barclay Stephens, San Francisco.—By invitation.

4. Acute Otitis Media—Indications for the Mastoid Operation.—Dr. Hill Hastings, Los Angeles.

5. The Status Lymphaticus with Particular Reference to Anesthesia in Tonsil and Adenoid Operations.—Dr. W. H. Roberts, Pasadena.

W. H. Roberts, Chairman.

Business Meeting.

At 3:30 p. m. the Society went into executive session.

The election of Fellows was the first order of business. The Secretary announced that the following candidates had been recommended by the Council for election:

John H. Allen, M. D., Portland, Maine. Proposed by D. B. Kyle, M. D., seconded by Irving E. Kimball, M. D.

William B. Chamberlin, M. D., Cleveland, Ohio. Proposed by J. M. Ingersoll, M. D., seconded by Wm. R. Lincoln, M. D.

Walter F. Chappell, M. D., New York City, N. Y. Proposed by Arthur B. Duel, M. D., seconded by Harmon Smith, M. D.

Percy Fridenberg, M. D., New York City, N. Y. Proposed by T. J. Harris, M. D., seconded by J. F. McKernon, M. D.

Harry Friedenwald, M. D., Baltimore, Maryland. Proposed by H. O. Reik, M. D., seconded by John R. Winslow, M. D.

Secord H. Large, M. D., Cleveland, Ohio. Proposed by Wm. L. Lincoln, M. D., Seconded by J. M. Ingersoll, M. D.

J. J. LaSalle, M. D., Toledo, Ohio. Proposed by Thomas Hubbard, M. D., seconded by J. M. Ingersoll, M. D.

John R. Page, M. D., New York City, N. Y. Proposed by Arthur B. Duel, M. D., seconded by Lewis A. Coffin, M. D.

Andrew J. N. Reik, M. D., Baltimore, Maryland. Proposed by H. O. Reik, M. D., seconded by John R. Winslow, M. D.

Sylvan Rosenheim, M. D., Baltimore, Maryland. Proposed by H. O. Reik, M. D., seconded by John R. Winslow, M. D.

A. John Smith, M. D., Yonkers, New York. Proposed by E. B. Dench, M. D., seconded by W. C. Phillips, M. D.

Alfred Wiener, M. D., New York City, N. Y. Proposed by Jos. H. Abraham, M. D., seconded by Lewis A. Coffin, M. D.

On motion, the Secretary was instructed to cast an affirmative ballot for the list of candidates, as read; which was done and they were declared duly elected.

The President then appointed as Auditing Committee the following: Drs. Duel, Beck and Gallaher.

On motion, the President appointed the following as a committee to serve as the Nominating Committee: Drs. Stucky, Leland, McKernon, Allport, and Pierce.

The Committee on Place of Meeting, through its Chairman, Dr. James F. McKernon, reported that Atlantic City had been selected as the place of meeting for 1909. On motion, this report was approved and accepted.

The Committee on Nomenclature, through Dr. McKernon, made the following report:

The Committee appointed at the Annual Meeting in 1907 to "Consider and Recommend a Suitable Nomenclature for use in

Affections of the Nose, Throat and Ear" respectfully offer the following report of progress.

Your Committee has carefully considered the subject, and believes that the following premises will be admitted by all.

Previous experience has shown that any scheme of nomenclature introduced by a comparatively small body of those interested in the subject fails to command the interest and co-operation even of all the members of that body.

A system of nomenclature in one department of medicine should as far as possible correspond with systems advocated and used in other departments of medicine.

In order to replace the present use of varying and often illogical terms which have by slow growth become a part of our language, by a system satisfactory to the largest number, the system must command the greatest possible respect. This can only be done by its endorsement by as large a part of the profession as possible, and by its inherent excellence. This necessarily will take much time and co-operation.

Your committee have deemed it unwise to prepare any scheme of nomenclature to be presented at the present meeting, as such a scheme would probably arouse more discussion or protest than could be satisfactorily answered at the meeting.

The members of your committee have been appointed as a Committee of the American Laryngological Association at its recent Annual Meeting to consider the same subject, in order to promote co-operation between the two societies.

It is probable that the American Medical Association will consider the question of nomenclature at its coming session, and it is possible that an extensive movement will shortly develop towards a rational and widely acceptable system.

If the society continue the life of the committee, it will endeavor to co-operate with similar committees and to harmonize our department with other departments of medicine.

W. Coolidge,
D. Braden Kyle,
Jas. F. McKernon.

It was moved and seconded that the report be accepted and the committee continued.

Dr. Phillips moved an amendment to this motion to the effect that the Society instruct the committee to meet with a similar committee from each of the following societies: American Laryngological Association, Laryngological and Otological Section of the American Medical Association, and the American Otological Society.

The motion as amended was carried.

The Society went into its second executive session at 10 A. M., May 29.

The report of the Secretary was read and approved.

Report of the Secretary.

The Secretary has to report the death since the last annual meeting of Dr. W. G. B. Harland of Philadelphia, Sept. 14th, 1907, and of Dr. D. B. St. John Roosa of New York, March 8th, 1908 Dr. Harland was elected in 1905 and Dr. Roosa in 1895.

The transactions of the thirteenth annual meeting were issued shortly after the 1st of February. It is a volume of over 550 pages and in every particular, reflects credit upon the Publication Committee and the publisher. The Committee has also at the request of the Council prepared a complete index by subjects and authors of the several volumes of the Transactions beginning with volume one. This should be in the hands of all the members by the present time. The meetings of the four Sections have been held and proved in point of attendance and of scientific worth, all that could be desired. The Council has held three meetings during the year. It is to be regretted that no theses have been received in competition for the prizes offered or for the Research Fund. There has been one resignation. The total membership including those elected at this meeting is 287 of whom 23 are Honorary Fellows.

Thomas J. Harris,
Secretary.

The Report of the Treasurer was next read. The Auditing Committee reported that the accounts had been examined and found to be correct.

TREASURER'S REPORT, MAY 31, '07, TO MAY 30, '08.

Balance from 1906-1907	$1,318.30
193 Current Dues, @ $10	1,930.00
49 Back Dues, @ $10	490.00
Allowed on Checks for Collection	.40
16 Initiation Fees, @ $10	160.00
25 Transactions, @ $2.50	62.50
1 Abstract and Discussion, @ $12	12.00
4 Killian Medals, @ $2.50	10.00
1 Note	500.00
Total Cash for 1907-1908	$4,483.26
Total Expense for 1907-1908	4,361.32
Balance	$ 121.94

ACCOUNTS DUE THE SOCIETY

Members in arrears for 1 year (43), @ $10 per year	$ 430.00
Members in arrears for 2 years (12), @ $10 per year	240.00
Total Outstanding Accounts	$ 670.00

DISBURSEMENTS BY VOUCHERS.

Two Bonds	$2,062.50	$2,062.50
Reporting and Trans. Meetings '06-'07	145.05	145.05
Section Meetings—		
Eastern Section	25.00	
Southern Section	25.00	
Middle Section	25.00	75.00
Publication Committee	50.00	50.00
Secretary's Expenses, '06-'07	117.80	
Secretary's Expenses, '07-'08	192.00	309.80
Lincoln Note and Interest	509.90	509.90
Deficit on Annual Dinner	100.00	100.00
Transactions, 1906	220.49	220.49
Manhattan Storage	340.11	340.11
Clerical Hire	33.02	
Engrossing Certificates	5.05	
Printing, Postage, Express, Telephone and Stationery	361.50	399.57
Forward		$4,222.32

Forward		$4,222.32
Deficit on Prof. Frankel's 70th Birthday Celebration	7.00	7.00
Prof. Killian's Demonstration	17.00	17.00
Hotel Astor for Prof. Killian	42.75	42.75
Deficit on Killian Medal and Electrotypes	41.00	41.00
Cable to Dr. Cheatle, London	10.75	10.75
Anatomical Specimens and Microscopes	18.00	18.00
Return Check to Dr. Harland	2.50	2.50
Total Expense for 1907-1908		$4,361.32
		121.94
		$4,486.26

CHEVALIER Q. JACKSON, Treasurer.

May 30th, 1908.

The above account audited and found correct.

ARTHUR B. DUEL,
JOS. C. BECK,
T. J. GALLAHER.
Auditing Committee.

The Nominating Committee reported as follows:

President.—Christian R. Holmes, Cincinnati, Ohio.

Vice-Presidents and Chairmen of Sections:

Eastern Section.—Francis R. Packard, Philadelphia, Pa.

Middle Section.—R. Bishop Canfield, Ann Arbor, Mich.

Western Section.—Joseph Beck, Chicago, Ill.

Southern Section.—John Dunn, Richmond, Va.

Secretary.—John F. Barnhill, Indianapolis, Ind.

Treasurer.—Ewing W. Day, Pittsburg, Pa.

Council.—George L. Richards, Fall River, Mass.; Thomas J Harris, New York City; G. Hudson Makuen, Philadelphia; William R. Lincoln, Cleveland, Ohio.

Library Committee.—Chairman, H. Holbrook Curtis, New York; Lewis A. Coffin, New York; Clement F. Theisen, Albany, N. Y.; Norton L. Wilson, Elizabeth, N. J.

Publication Committee.—William H. Haskin, New York; Otto J. Stein, Chicago; Thomas H. Halsted, Syracuse, N. Y.

On motion, the Secretary was instructed to cast the ballot of the Society for the officers whose names had been duly presented, which he did, and they were declared elected.

The Library Committee, through its Chairman, Dr. H. Holbrook Curtis, rendered a report of the committee's work for the year.

Dr. Phillips, in moving that the report be accepted, also moved that the Society recommend to the Council that sufficient appropriation be made to carry on the work of the Library Committee. Seconded and carried.

The Committee appointed for the purpose of investigating the advisability of the Society publishing an official journal, through its Chairman, Dr. E. Fletcher Ingals, rendered the following report, which was freely discussed:

At the last meeting of the American Laryngological, Rhinological and Otological Society, Drs. Dench, Richardson and myself, were appointed as a Committee to consider the subject of having that Society, the American Laryngological Association and the American Otological Society publish a Journal covering the three subjects and containing their transactions..

We have gone over the matter very thoroughly and beg to report that the publication of such a Journal appears to us feasible providing it is made of high order and is supported by the three Societies.

In our opinion if a Journal is published it should be an authoritative exponent of the progress of medical science in laryngology, rhinology and otology.

It should contain: *First*, original articles of high order either of clinical or laboratory research work on the principles and practise of our specialties. *Second.* (a) Concise but lucid abstracts of the papers and discussions presented at the various meetings of these three organizations.

(b) Abstracts of all the valuable current literature on these subjects throughout the world (provided the expense of abstracting would not be too great).

(c) The official business transactions of each organization.

(d) Ethical advertisements.

It may seem desirable that this Journal be published in two parts, like the Journal of Anatomy, each of which should be paged separately and possibly printed in different type. The second part of the Journal might be in smaller type and contain the transactions of the three Societies and thus take the place of the official transactions.

The Journal should be printed on excellent paper, in good style. It should be printed from good type and should be carefully edited.

The Journal of Anatomy or the Archives of Otology give a fair idea of our conception of such a Journal.

It should be published monthly.

It should be paid for pro rata by the various Societies according to their active membership, one copy to be sent to each active member of these Societies, for which he should be assessed $5.00 per year by whichever Society he might elect. This amount is an addition to the regular annual dues.

As this plan would allow the accummulation of funds in the treasury of the various societies that would otherwise be expended for publication of their transactions, it is thought by the Committee that these funds could be used to cover any deficit which might occur during the first years of the publication of such a Journal.

There should be one responsible editor with two or three associates and with all needed collaborators.

The Journal should be controlled by a Board of Trustees of one or two members from each of the Societies; these Trustees to be appointed by the Society that they represent. The Board of Trustees should have complete control of the financial and other interests of the Journal. They should appoint the editor-in-chief, and should, if they thought best, appoint also a first-class manager. The Board of Trustees with the consent of the councils of the various Societies should determine what financial obligations might be incurred.

The editor-in-chief should appoint the associates by advice and consent of said Trustees, and he should be responsible for all editorial work on the Journal and for the ethical standard of the advertisements. The business manager to be appointed by the Board of Trustees might or might not be one of the editors, according to the judgment of the Trustees. He should be responsible to the Trustees but subject to the editor-in-chief in all matters of ethics. The Journal should be published by some first-class publishing house that would do all of the work connected with publication and circulation.

Such a publication appears to your Committee and to many of our fellows as very desirable. Your Committee, however, dislikes the multiplication of Journals, therefore urges the combination or all existing interests that can be acquired without expense to any of these organizations that would be consistent with the high character of the proposed archives. It appears to your Committee that the

work of the editors would, at least at first, have to be rendered gratuitously, but that ultimately it should be paid for, provided the Journal became a financial success. The business manager should receive a small compensation for his services, which might be made, partly at least, dependent upon his success in securing advertisements.

E. Fletcher Ingals.
Edward B. Dench.
Chas. W. Richardson.

The Secretary reported that the Council had carefully considered the matter under discussion, and felt confident that the best interests of the Society would be conserved by the Committee.

It was then moved, seconded and carried that the matter of the journal be left with the Committee, with power to act.

The Secretary reported the resignation of Dr. Arnold Knapp, which, on motion, was accepted, with regrets.

On motion, duly seconded and carried, a vote of thanks was extended to Dr. William H. Haskin, Chairman of the Publication Committee, for his excellent work.

The Secretary reported that the Council desired an expression from the Society with reference to the advisability of placing a limitation upon the membership. After very free and liberal discussion, it was moved and seconded that the Society limit its membership. Motion lost.

The Secretary read a further recommendation from the Council to the effect that any member who fails to attend one general meeting of the Society in three years be dropped from membership. After full discussion of the recommendation it was moved, seconded and carried, that if any member of the Society fails to attend either an annual meeting or a section meeting once in three years, the matter shall be taken up by the Society for action, this ruling to go into effect three years hence.

It was moved and seconded and carried that from this time the issuing of certificates of election be discontinued.

It was moved, seconded and carried, that the Secretary be instructed to send to the members of the Society a notice of the resolution concerning the dropping of members.

The President read a resolution from the Council to the effect that all papers presented before the Society are the property of

the Society, for its exclusive use, to be published elsewhere than in the transactions only upon consent of the Society.

It was moved, seconded and carried, that Professor Jansen be made an Honorary Fellow of the Society.

At the close of the scientific session, May 30, the Society went into executive session.

Dr. John F. Barnhill, the newly elected Secretary, offered his resignation from this office, and named as his successor Dr. Thomas J. Harris. On motion, duly seconded and carried, Dr. Barnhill's resignation was accepted and Dr. Harris elected in his place. It was then moved, seconded and carried that Dr. Barnhill be made a member of the Council, vice Dr. Harris.

On motion a vote of thanks was extended to the retiring President and other Pittsburg members for their hospitality and for their many courtesies during the meeting.

To the Council:

As Chairman of the Publication Committee I have the honor to report that, thanks to the very thoughtful co-operation of Dr. M. A. Goldstein, we were enabled to issue the volume by February 1st, 1908, and had we been able to secure all of the papers on time it would have been issued during December. I think that the Society owes Dr. Goldstein a vote of thanks for the splendid preparation of the volume, which our Committee feels is the best that has yet been published.

Respectfully submitted,

W. H. Haskin,

Chairman.

The newly elected President, on taking the Chair, expressed his sincere appreciation of the honor conferred upon him by the Society.

There being no further business, the Society adjourned.

FELLOWS.

Abraham, Jos. H.....616 Madison Ave., New York
Abrams, Alva E.....78 High St., Hartford, Conn.
Adams, John L.....38 East 51st St., New York
Alderton, Henry A.....142 Clinton St., Brooklyn, N. Y.
Allen, John H.....111 Congress St., Portland, Me.
Allport, Frank A.....72 E. Madison St., Chicago, Ill.
Anderson, Willis S.....606 Washington Arcade, Detroit, Mich.
Ard, F. C.....604 Park Ave., Plainfield, N. J.
Arnold, J. Dennis.....1296 Van Ness Ave., San Francisco, Cal.
Arrowsmith, Hubert .....180 Clinton St., Brooklyn, N. Y.
Atkinson, Wm. J.....111 North Main St., Paterson, N. J.
Babbitt, Jas. A.....112 South 18th St., Philadelphia, Pa.
Ballenger, Wm. L.....100 State St., Chicago, Ill.
Bane, Wm. C.....Commonwealth Bldg., Denver, Colo.
Barnhill, John F.....Indianapolis, Ind.
Beck, Jos.....100 State St., Chicago, Ill.
Berens, Bernard .....2041 Chestnut St., Philadelphia, Pa.
Berens, T. Passmore .....35 Park Ave., New York
Birkett, H. S.....252 Mountain St., Montreal, Can.
Blake, Clarence J.....226 Marlboro St., Boston, Mass.
Booth, Burton S.....21 First St., Troy, N. Y.
Borden, Charles R. C.....223 Newberry St., Boston, Mass.
Braislin, W. C.....556 Washington Ave., Brooklyn, N. Y.
Brown, J. Price .....37 Carlton St., Toronto, Ont., Can.
Bryant, Wm. Sohier .....57 West 53d St., New York
Burnett, Peter V.....170 Keap St., Brooklyn, N. Y.
Calhoun, A. W.....62 Marietta St., Atlanta, Ga.
Campbell, Wm. E.....606 Century Bldg., Atlanta, Ga.
Canfield, R. Bishop .....Ann Arbor, Mich.
Chamberlain, W. B.....318 Euclid Ave., Cleveland, O.
Chambers, Talbot R.....490 Jersey Ave., Jersey City, N. J.
Chappell, Walter F.....7 East 55th, New York

Cheatham, Wm.....303 West Chestnut St., Louisville, Ky.
Chenery, Wm. E.....222 Huntington Ave., Boston, Mass.
Chisholm, W. Alexander .....236 W. 44th St., New York
Christy, T. C.....717 Westinghouse Bldg., Pittsburg, Pa.
Clark, Jos. Payson .....409 Marlboro St., Boston, Mass.
Clemens, Jas. B.....15 East 48th St., New York
Cline, L. C.....224 North Meridan St., Indianapolis, Ind.
Coakley, C. G.....53 West 56th St., New York
Coates, Geo. M.....334 South 19th St., Philadelphia, Pa.
Cobb, Frederick C.....11 Marlboro St., Boston, Mass.
Coffin, Lewis A.....156 West 58th St., New York
Coggeshall, Henry .....40 East 58th St., New York
Cohen, J. Solis .....1824 Chestnut St., Philadelphia, Pa.
Collins, Burnett .....645 St. Marks Place, Brooklyn, N. Y.
Connell, J. C.....265 King St., Kingston, Ont., Can.
Corwin, Theodore .....5 West Park St., Newark, N. J.
Coolidge, A.....613 Beacon St., Boston, Mass.
Cott, Geo. F.....85 North Pearl St., Buffalo, N. Y.
Cox, Chas. N.....257 Jefferson Ave., Brooklyn, N. Y.
Craig, Robert H.....186 Peel St., Montreal, Can.
Crockett, Eugene A.....298 Marlboro St., Boston, Mass.
Culbert, Wm. Ledlie .....54 East 34th St., New York
Culp, John F.....Harrisburg, Pa.
Curtis, H. Holbrook .....118 Madison Ave., New York
Dabney, Wm. R.....Marietta, Ohio
Day, Ewing W.....Westinghouse Bldg., Pittsburg, Pa.
Daly, Walter S.....Ogdensburg, N. Y.
Dench, Edward B.....17 West 46th St., New York
Dessar, L. A.....156 West 58th St., New York
Donnellan, Patrick S.....1028 Spruce St., Philadelphia, Pa.
Dudley, Wm. F.....32 Livingston St., Brooklyn, N. Y.
Dudley, Wm. H.....Los Angeles, Cal.
Duel, Arthur B.....254 Madison Ave., New York
Dunn, John .....314 East Franklin St., Richmond, Va.
Dye, Hobart S.....1404 L St., N. W., Washington, D. C.
Eagleton, Wells P.....15 Lombardy St., Newark, N. J.
Ellis, H. Bert.....245 Bradbury Bldg., Los Angeles, Cal.
Emerson, Francis P.....404 Boylston St., Boston, Mass.
Emerson, Linn .....Orange, N. J.
Farrell, Thomas H.....236 Genesee St., Utica, N. Y.

Fetterolf, Geo.....328 South 15th St., Philadelphia, Pa.
Fitzpatrick, T. V.....26 Garfield Place, Cincinnati, Ohio
Fleming, Ernest W.....214 Bradbury Block, Los Angeles, Cal.
Foster, John M.....11 Stedman Bldg., Denver, Colo.
Freeman, Walter J.....1720 Locust St., Philadelphia, Pa.
Freudenthal, Wolff .....1003 Madison Ave., New York
Friedenwald, Harry .....1029 Madison Ave., Baltimore, Md.
Fryer, Blencowe E....Cor. 9th and Cherry Sts., Kansas City, Mo.
Gallaher, Thomas J.....California Bldg., Denver, Colo.
Gibb, Jos. S.....1907 Chestnut St., Philadelphia, Pa.
Gildea, Patrick F.....2 Cascade Ave., Colorado Springs, Colo.
Goff, Waldo P.....Clarksburg, W. Va.
Goldsmith, Perry G.....Belleville, Ont.
Goldstein, Max A.....3858 Westminster Place, St. Louis, Mo.
Gradle, Henry .....100 State St., Chicago, Ill.
Graef, Charles .....1076 Boston Road, New York
Grayson, Chas. P.....251 South 16th St., Philadelphia, Pa.
Greene, D. C., Jr.....12 West Cedar St., Boston, Mass.
Griffin, Edwin H.....112 West 45th St., New York
Gruening, Emil .....35 East 57th St., New York
Haddock, Chas. W.....14 Bartlett St., Beverly, Mass.
Halsted, Thomas H.....831 University Block, Syracuse, N. Y.
Hall, D. Walton .....Bryant Bldg., Kansas City, Mo.
Harris, Thomas J.....117 East 40th St., New York
Hartz, Henry J.....27 Adams St., East Detroit, Mich.
Haskin, Wm. H.....42 East 41st St., New York
Hastings, Hili .....Los Angeles, Cal.
Hedges, Halsted S.....Charlotteville, Va.
Held, R. Johnson .....616 Madison Ave., New York
Hinkel, F. W.....412 Franklin St., Buffalo, N. Y.
Holmes, Christian R.....8 East 8th St., Cincinnati, Ohio
Holmes, Edgar M.....531 Beacon St., Boston, Mass.
Hopkins, F. E.....25 Harrison Ave., Springfield, Mass.
Hopkins, Wm. F.....803 Sutter St., San Francisco, Cal.
Hubbard, Dwight L.....135 West 94th St., New York
Hubbard, Thomas .....205 Ontario St., Toledo, Ohio
Hurd, Lee M.....15 East 48th St., New York
Hyatt, Frank .....1022 14th St., N. W., Washington, D. C.
Ingals, E. Fletcher .....34 Washington St., Chicago, Ill.
Ingersoll, John M.....318 Euclid Ave., Cleveland, Ohio

Jack, F. L.....215 Beacon St., Boston, Mass.
Jackson, Chevalier .....428 Park Bldg., Pittsburg, Pa.
Jackson, Oliver H.....34 North Main St., Fall River, Mass.
Joachim, Otto .....501 Marecha Bldg., New Orleans, La.
Jones, Clarence Porter .....Newport News, Va.
Keiper, Geo. F.....Lafayette, Ind.
Kenefick, Jos. A.....78 E. 54th St., New York
Kennon, Beverley R.....64 Granby St., Norfolk, Va
Kent, J. L.....1101 Church St., Lynchburg, Va
Kerrison, Philip D.....58 West 56th St., New York
Kimball, Irving E.....717 Congress St., Portland, Me
Kirkendall, J. S.....Ithaca, N. Y.
Knowles, W. F.....220 Clarendon St., Boston, Mass.
Kopetzky, Samuel J.....616 Madison Ave., New York
Koyle, F. H.....199 Main St., Hornellsville, N. Y.
Kuyk, D. A.....Richmond, Va.
Kyle, D. Braden .....1517 Walnut St., Philadelphia, Pa.
Kyle, John J.....The Newton Claypool Bldg., Indianapolis, Ind.
Large, Secord H.....536 Rose Bldg., Cleveland, Ohio
Laselle, James J.....226 Michigan St., Toledo, Ohio
Lederman, M. D.....58 East 75th St., New York
Leland, Geo. A.....354 Commonwealth Ave., Boston, Mass.
Leonard, Z. L.....19 West 120th St., New York
Lester, John C.....179 Schermerhorn St., Brooklyn, N. Y.
Levy, Robert .....300 California Bldg., Denver, Colo
Lewis, Robert, Jr.....48 West 40th St., New York
Lincoln, Wm. R.....Lenox Bldg., Cleveland, Ohio
Loeb, H. W.....537 North Grand Ave., St. Louis, Mo.
Logan, Jas. E.....1208 Wyandotte St., Kansas City, Mo.
Lowman, John B.....142 Park Place, Johnstown, Pa.
Lutz, Stephen H.....284 Hancock St., Brooklyn, N. Y.
Mahon, John S.....721 Sandusky St., Allegheny, Pa.
Macpherson, D.....467 Manhattan Ave., New York
Makuen, G. Hudson .....1627 Walnut St., Philadelphia, Pa.
Malcolm, Percy E. D.....55 W. 55th St., New York
Mason, Wm. Beverly...812 Connecticut Ave., Washington, D. C.
Means, Chas. S.....715 North High St., Columbus, Ohio
Mial, Leonidas L.....133 West 11th St., New York
Miller, Frank E.....22 West 31st St., New York
Miller, Lewis H.....287 Clinton Ave., Brooklyn, N. Y.

Milligan, Robert ....Smith Block, 6th and Liberty, Pittsburg, Pa.
Mooney, Jas. J.....393 7th St., Buffalo, N. Y.
Moore, Thomas W.....1048 Third Ave., Huntington, W. Va.
Morgenthau, Geo.....34 Washington St., Chicago, Ill.
Mosher, Harris P.....828 Beacon St., Boston, Mass.
Moss, Robert E.....Hicks Bldg., San Antonio, Tex.
Mullins, John B.....1516 H St., Washington, D. C.
Munger, Carl E.....Waterbury, Conn.
Murphy, John W.....4 West 17th St., Cincinnati, Ohio
Murray, Gilbert D.....436 Wyoming Ave., Scranton, Pa.
Myers, Henry L.....151 Branby St., Norfolk, Va.
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McGahan, Chas. F.....Aiken, S. C.
McKernon, Jas. F.....62 West 52d St., New York
McKimmie, O. A. M..1330 Mass. Ave., N. W., Washington, D. C.
McLaury, Frank H.....551 West 161st St., New York
McReynolds, John O.....Frost Bldg., Dallas, Tex.
Norris, C. W.....Lexington, Ky.
Off, Henry J.....121 South 18th St., Philadelphia, Pa.
O'Kelly, Jas. P.....501 Macheca Bldg., New Orleans, La
Oppenheimer, Seymour .....45 East 60th St., New York
Packard, Francis R.....19th and Pine Sts., Philadelphia, Pa.
Page, John R.....254 Madison Ave., New York
Park, J. Walter .....32 North 2d St., Harrisburg, Pa.
Parker, Edward F.....70 Hassell St., Charleston, S. C.
Patterson, Jas. A....805 North Trejon St., Colorado Springs, Colo.
Payne, Redmond W.....915 Van Ness Ave., San Francisco, Cal
Peete, C. H.....Macon, Ga.
Phillips, Wendell C.....40 West 47th St., New York
Pierce, Norval H.....31 Washington St., Chicago, Ill.
Pischel, Kaspar .....1817 California St., San Francisco, Cal
Powell, Stephen C.....Newport, R. I.
Quinlan, F. J.....33 West 38th St., New York
Rae, John B.....616 Madison Ave., New York
Ray, J. M.....432 West Chestnut St., Louisville, Ky.
Reik, Andrew J. N.....412 Cathedral St., Baltimore, Md.

Reik, Henry O.....412 Cathedral St., Baltimore, Md.
Renner, W. Scott .....361 Pearl St., Buffalo, N. Y.
Reuling, Geo.....103 West Monument St., Baltimore, Md.
Richards, George L.....84 North Main St., Fall River, Mass.
Richards, John D.....44 West 49th St., New York
Richardson, Chas. W.....1317 Connecticut Ave., Washington, D. C.
Roberts, Walter .....33 South 19th St., Philadelphia, Pa.
Roberts, W. Humes .....Pasadena, Cal.
Robinson, John A.....209 West 85th St., New York
Roe, John O.....28 Clinton Ave., N. Rochester, N. Y.
Rogers, Frederick T.....117 Broad St., Providence, R. I.
Root, Arthur G.....218 State St., Albany, N. Y.
Rosenheim, Sylvan .....1710 Linden Ave., Baltimore, Md.
Ross, Geo. T.....945 Dorchester St., Montreal, Can.
Roy, Dunbar .....Grand Opera House Block, Atlanta, Ga.
Sandles, C. C.....Westinghouse Bldg., Pittsburg, Pa.
Sauer, Wm. E.....Grand and Washington Aves., St. Louis, Mo.
Schadle, J. E.....140 Lowry Arcade, St. Paul, Minn.
Seiss, Ralph W.....213 South 17th St., Philadelphia, Pa.
Sharp, J. Clarence .....42 West 46th St., New York
Shattuck, Warren S., Jr.....147 Clinton St., Brooklyn, N. Y.
Sheppard, J. E.....130 Montague St., Brooklyn, N. Y.
Shorter, J. H.....Macon, Ga.
Shields, Wm. B.....303 North Grand Ave., St. Louis, Mo.
Shurly, B. R.....32 West Adams Ave., Detroit, Mich
Smith, A. John .....Yonkers, N. Y.
Smith, Harmon .....44 West 49th St., New York
Smith, S. MacCuen .....1700 Walnut St., Philadelphia, Pa.
Smith, W Harvey .....250 Donald St., Winnipeg, Manit.
Smith, E. Terry .....75 Pratt St., Hartford, Conn.
Smyth, Herbert E.....376 John St., Bridgeport, Conn.
Snow, Sargent F.....707 University Block, Syracuse, N. Y.
Solenberger, Amos R...106 E. St. Vrain Ct., Colo. Springs, Colo.
Sondern, Paul F.....52 South Fitzhugh St., Rochester, N. Y.
Sprague, Frank B.....27 Stewart St., Providence, R. I.
Spratt, Chas. Nelson..310 Syndicate Arcade, Minneapolis, Minn.
Steel, Geo. E.....124 West 119th St., New York
Stein, Otto Jacob .....100 State St., Chicago, Ill.
Sterrett, John Kennedy .....320 Smith Block, Pittsburg, Pa.
Stillman, F. L.....118 East Broad St., Columbus, Ohio

Stout, Geo. C.....34 South 18th St., Philadelphia, Pa.
Stucky, J. A.....The McClelland Bldg., Lexington, Ky.
Theisen, Clement F.....172 Washington Ave., Albany, N. Y.
Thigpen, C. A.....13 South Ferry St., Montgomery, Ala.
Thigpen, F. M.....Pensacola, Fla.
Thompson, John A.....628 Elm St., Cincinnati, Ohio
Thompson, J. C.....302 Ninth Ave., Pittsburg, Pa.
Thomson, John J.....40 West 47th St., New York
Thrasher, A. B.....N. E. Cor. 7th and Race Sts., Cincinnati, Ohio
Thrasher, Wade....N. E. Cor. 7th and Race Sts., Cincinnati, Ohio
Trowbridge, D. H.....Fresno, Cal.
Turnbull, Thomas .....835 Wheaton Ave., Allegheny, Pa.
Tyson, Henry Hawkins .....47 West 51st St., New York
Vail, Derrick T.....24 East 8th St., Cincinnati, Ohio
Vansant, Eugene L.....1929 Chestnut St., Philadelphia, Pa.
Voislawsky, A. P.....128 West 59th St., New York
Voorhees, Sherman .....408 North Main St., Elmira, N. Y.
Wagner, Henry L.....506 Sutter St., San Francisco, Cal.
Wales, Ernest DeW....320 North Meridian St., Indianapolis, Ind.
Waterman, Jas. S.....676 St. Marks Ave., Brooklyn, N. Y.
Webster, Geo. A.....419 Boylston St., Boston, Mass.
Wells, Walter A.....1113 14th St., Washington, D. C.
White, Joseph A.....200 East Franklin St., Richmond, Va.
Wiener, Alfred .....616 Madison Ave., New York
Wilson, Norton L.....410 Westminster Ave., Elizabeth, N. J.
Winslow, John R.....114 Franklin St., Baltimore, Md.
Wishart, D. J. Gibb.....47 Grosvenor St., Toronto, Can.
Wood, George B.....129 South 18th St., Philadelphia, Pa.
Woodward, John F.....Norfolk, Va.
Yates, D. G.....79 West 104th St., New York

HONORARY FELLOWS.

Ballance, C. A.....London, England
Gradenigo, G.....Turin, Italy
Grant, Dundas .....London, England
Herying, T.....Warsaw, Russia
Jansen, Albert .....Berlin, Germany
Karwowski, Constantine .....Warsaw, Russia
Killian, G.....Freiburg, Germany
Law, Edward .....London, England

Luc, H.....Paris, France
MacIntyre, John .....Glasgow, Scotland
Martin, A.....Paris, France
Moure, E. J .....Bordeaux, France
Martinez, Emilio .....Havana, Cuba
Onodi, Adolph .....Budapest, Hungary
Politzer, A.....Vienna, Austria
Poyet, George .....Paris, France
Semon, Sir Felix .....London, England
Schmiegelow, E.....Copenhagen, Denmark
Sendziak, J.....Warsaw, Russia
Turner, Logan .....Edinburgh, Scotland
Woakes, W.....London, West, England
Wolfenden, R. Morris .....London, England
Ziem, C. H.....Danzig, Germany

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